

The Treatment of
INJURIES TO
THE NERVOUS SYSTEM

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AFFECTIONATELY DEDICATED
TO
M H L AND M F M



Preface

This book is written primarily for the use of the general surgeon and the general practitioner. It has been obvious for a long time that the bulk of neurologic surgery lies in the field of trauma and not, as used to be the case in the field of tumors. The corollary to this must be that the bulk of neurosurgery will be seen first by the general surgeon and the general practitioner. In the course of more than twenty years of neurosurgical practice it has become apparent to me that these men have been greatly handicapped in their care of these patients by the lack of any concise source of information. The therapy of injuries to the nervous system is so new and is still in such a state of flux—particularly that pertaining to the spinal cord and cauda equina—that, while such information is procurable from the medical journals, it has not yet been made available in a more compact form. Moreover, lack of knowledge as to modern therapeutic methods is not the only handicap under which the general surgeon works. He has also no concept of what end-results he and his patient should legitimately expect from his own or his neurosurgical consultants' ministrations. This aspect of the neurosurgical problem was forcibly brought to my attention by Mr. Stanwood L. Hanson, who has so kindly contributed the chapter *The Cost of Medical Care in Paraplegia and How It May be Modified by Rehabilitation Services*. He served in addition and without his knowledge as the spark plug which motivated this book but he should not be held responsible for any but the good parts of it.

In this volume are the methods that have been evolved for and are now in daily use in the treatment of all types of injury to the nervous system at my neurosurgical clinic at the Boston City Hospital. The procedures described are by no means the only methods and are not even necessarily the best methods for the treatment of the conditions that are considered. They do, however, have the virtues of practicality, the authority that comes from evolutionary development over a number of years, the stability that is developed only with use by a number of different members of the staff, and the honesty that is inherent in a constant checking of mistakes by the house staff and by postmortem examinations made by or under the direction of a medical examiner.

The work done in other clinics and by other neurosurgeons has been drawn upon freely and adapted to our needs but, much as I would like to do so, it is impossible to credit these benefactors in detail. Reference to such work, therefore, has been made only when I or the members of my clinic have had no personal experience with the methods cited. In addition I should like to take this opportunity to add my word of appreciation and thanks to

some whose names do not appear in the bibliography. Their help has been given in fields larger than those of specific methods, although here too I have taken advantage of their experience and knowledge. They have contributed, rather that intangible factor which serves to encourage others in their difficult moments. This has been particularly true in my work with the paraplegics and quadriplegics. Leading this list are Mr John G Counsell, O.B.E. M.C., presently president of the Canadian Paraplegic Association and himself a paraplegic, and his medical colleague Dr E. H. Bottrell, O.B.E., M.D., M.S., F.R.C.S (C) of Toronto. To these men should go the credit for really setting in motion the rehabilitation of the spinal cord casualties of World War II. Similar contributions in this country have been made by Miss Betsy Barton—also a paraplegic—by Dr Loyal Davis and his group at Hines Veterans Administration Hospital, and by Dr Ernest Bors of California. In the more restricted field of physical rehabilitation I like all others interested, have greatly profited by the advances first sparked by Dr George G Deaver and his associates Misses Brown and Buchwald, and then raised to previously unimagined heights by the efforts of Messrs. E. M. Saunders and Alfred B. Ellison and their assistants.

In my own clinic many have been of inestimable help. My two associates—Dr Walter R. Wegner and Dr A. Price Heuser—the many residents, house-officers, head-nurses and other nurses cannot be thanked enough. The one among the house-staff and nurses to whom I owe most, however is Miss Jane Dignan presently a teaching supervisor at the Boston City Hospital. Her contributions in time spent, interest taken, sacrifices made and professional acumen brought to bear on the problems of nursing care as well as the physical and social rehabilitation of patients with injuries to their nervous systems cannot be recompensed.

My thanks are due to Dr Donald Bickers, whose text and illustrations I have used in the section on Splinting for Ambulation to Dr Raymond D. Adams for lending me the splendid illustrations used in Figures 1 through 6 and to the New England Journal of Medicine for permission to reprint and use certain of the illustrations from the papers *Orthopedic Appliances in The Rehabilitation of Patients with Spinal Cord Injuries* (including Figures 1 through 7) volume 238 pages 545 to 553 issue of April 15 1948 written by Donald S. Bickers, and *The Rehabilitation of Patients Totally Paralyzed below the Waist, with Special Reference to Making Them Ambulatory and Capable of Earning Their Living. III Tidal Drainage, Cystometry and Bladder Training* (including Figures 2 through 8) volume 236 pages 223 to 235 issue of February 13 1947 written by me.

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The Central Nervous System

GENERAL CONSIDERATIONS

It is well to emphasize because of the frequency of occurrence and the practical considerations involved, certain of the more important points that must be faced at one time or another by all physicians in dealing with accidents and injuries that affect the skull, spine, brain spinal cord and cauda equina. Unfortunately such injuries are so common as to constitute a major problem. Their nature, moreover is such that if the best results are to be attained certain specialized care must be provided during both acute and chronic stages. The pathology varies from the simplest type for which no therapy is needed to that requiring the highest degree of surgical skill and judgment.

Causes

The causes of these injuries are well known and require only brief mention. In general they fall into such groups as accidents caused by moving vehicles falls, including diving accidents blows on the head and back from falling or moving objects and as the result of an attack and gunshot and stab wounds.

Transportation

Certain problems are common to all central-nervous-system injuries. The first of these has to do with the decisions that must be made concerning the need, the feasibility and the methods of transportation of the patient. It is axiomatic that severe surgical shock, under ordinary circumstances contra-indicates moving any patient. This applies to injuries of the central nervous system exactly as it does to any other injury. However after shock has been treated, if necessary on the spot, and the patient's blood pressure is satisfactory experience has shown that anyone suffering from a craniocerebral injury may be safely transported for any reasonable distance by ambulance, train or airplane providing normal atmospheric pressure is maintained. It is in the patient's best interests therefore to move him at once to a properly equipped and staffed hospital, even though this may involve a longer ride. Associated injuries to extremities should be splinted on the spot before the patient is moved. Excitement or major unco-operation at this time should,

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if possible be handled without artificial restraint, and morphine should never be given under such circumstances if it is possible to avoid it.

Transportation in the case of actual or potential spinal-cord injuries is much more dangerous. These patients must be subjected to a minimum of handling, and under no circumstances should any part of them except the arms be lifted off the ground, except as noted below. It is essential that they be rolled if further or new cord damage is to be avoided. Patients must be carried face down when transported, regardless of what level of the cord is involved. The litter or its substitute must be firm longer than the patient and of sufficient width to prevent his falling off. Any vehicle used must be a proper place for carrying such a patient. These patients may be transported also by train or airplane if strapped to a litter and accompanied by a trained attendant. Injuries to the cervical spine should be splinted temporarily and prior to moving by wrapping a thickly folded newspaper or a folded turkish towel of appropriate width around the neck in such a way as to support the head in slight extension on the clavicles and shoulders. The wrappings may be held in place with by emergency splinting and appropriate dressings on the spot. In a combined head and-spine injury the requirements for the spinal part take precedence. Unless and until the above criteria can be met, all patients with actual, potential or possible spine or spinal-cord injuries are much safer if they are left where they were found until they can be moved properly. They should be protected, of course by wrapping with sufficiently warm coverings against the weather and by guards against approaching traffic. Transportation to a hospital that is sufficiently metropolitan to meet the multiple demands of the neurosurgical, dietetic, genitourinary orthopedic, physiotherapeutic and possibly plastic surgical needs that these paralytics require should be provided as soon as possible after the accident, however if the rehabilitation that these patients now have the right to expect is to be provided for them. Less harm will be done—provided they are properly handled—by moving them once to their final hospital destination, even though it is more distant, than by moving them several times for shorter distances and thus necessitating more frequent handling.

Care of Open Wounds

The methods that are appropriate for surgical care of open wounds associated with trauma to the central nervous system are now well recognized and established. Primary dressings should be the simplest possible with a minimum of wound toilet and tissue manipulation. Chemotherapy should be administered in moderation, and antibiotics should be placed in the wound and given parenterally. Mechanical cleansing should not be undertaken until after the patient has reached the hospital, is out of shock, and whole blood transfusion is available. At that time a formal cleansing should be carried out, followed by débridement with particular regard to the individual layers of tissue and to the extraction of indriven bone fragments, and the gentle re

removal of all macerated brain tissue. Bullets should be removed only if easily accessible. Débridement of the spinal cord itself should not be attempted. Chemotherapy and appropriate antibiotics may if necessary be given intrathecally and if indicated, injected into the ventricles. Moreover it must be constantly borne in mind that it is not the sulfadiazine or penicillin that changes a contaminated or infected wound into one that is surgically clean but rather the surgical skill that is exercised in doing the débridement. Such definitive measures must if the patient's condition makes it possible be carried out within forty-eight hours of the receipt of the wound if a clean healing by first intention is to be expected. If the wound has been manipulated and the tissues mauled at the first dressing it may be necessary to carry out definitive therapy even before that time but this should not be done while the patient is in surgical shock for there is little to be gained by substituting a dead patient with a clean wound for a live one with a dirty wound.

Other Surgical Emergencies

Acute surgical emergencies such as intra abdominal hemorrhage a ruptured viscus, a tension pneumothorax or a sucking wound of the chest with hemopneumothorax, as well as the débridement of large destructive soft tissue wounds, must be dealt with before any neurologic surgery is performed because the former cannot and the latter may not be able to wait. Treatment of compound fractures of the extremities or of wounds of the joints may have to be postponed until the greater neurosurgical emergency has been met, however.

The Problem of Micturition

Severe injuries of the central nervous system cause some degree of atony or at least failure of the urinary bladder to empty. This applies to both brain and cord injuries although it is more severe and more constant in the cord injuries. Overflow incontinence should not be mistaken for normal micturition, nor can a full bladder necessarily be recognized by either palpation or percussion of the lower abdomen. Not only is it of the utmost importance to prevent overstretching of the bladder by the distention that accompanies the overflow incontinence of cord injuries, but the therapy needed in handling a patient with a contused or lacerated brain can also be greatly facilitated if his restlessness is diminished or possibly eliminated entirely by catheterization. The catheter should be whistle tipped and never larger than No. 14 French. Unless it is definitely known that conscious urinary control is present, sterile catheterization should be done as soon as possible after the patient's admission to the hospital. In unconscious, unco-operative and paraplegic or quadriplegic patients the catheter should be left in place and attached to a tidal-drainage apparatus as soon as possible (page 84). A solution of one half of 1 per cent acetic acid or 1:20,000 aqueous Zephiran should be used as irrigating fluid. Salt, boric acid and potassium permanganate solutions are contraindicated as irrigating media—the first because it causes the secretion of mucus the second, because it forms gravel and the

third because it is alkaline. Suprapubic cystostomy should never be performed. It is not even justified in combat zones, and when used there or if used under other conditions is less efficient as a method of drainage, slows up recovery, often leads to the need for later surgical closure and greatly increases the incidence of genitourinary-tract infection. The same objections apply to perineal urethrostomy. This moreover is much more difficult to close and when closed leaves a stricture of the urethra that would not otherwise have developed.

Bowels

Care of the bowel after surgical shock has subsided requires no unusual measures except in the case of major cord injuries (page 124). Enemas may be used as indicated, as may mineral oil and mild cathartics. Some attempt should be made to require the patient to attempt to move his bowels at the same hour every day in order that a proper bowel habit may be established as early as possible. One procedure should be avoided at all costs in the acute stage—that is, a laparotomy for supposed intestinal obstruction or retroperitoneal hemorrhage in the presence of a major spinal-cord injury. Actually the pathology is such that laparotomy only makes bad matters worse because the fundamental cause of the signs and symptoms is not obstruction or hemorrhage but a generalized paralytic ileus traceable to spinal shock. The proper treatment under such conditions is the withdrawal of all food and fluids (including water) by mouth and the intermittent use of a rectal tube until peristalsis can again be heard in the abdomen. In severe cases and especially in those patients with dilated stomachs this therapy should be supplemented by Wangensteen suction drainage.

Fluids

The administration of sufficient amounts of fluid preferably by way of the stomach is a *sine qua non* in the treatment of both brain and cord injuries. Provided that the patient is an adult and has a compensated heart and a normal genitourinary tract and is not debilitated because of advanced age or arteriosclerosis, one who has sustained an injury to the central nervous system should receive in addition to that in his diet, a minimum of 3600 cc of fluids every twenty-four hours. If these prerequisites cannot be met or if edema of the extremities, back or lungs develops, this minimal amount should be scaled down to an appropriate figure. When there has been a great quantity of body fluids lost through vomiting, excessive sweating, high temperature, increased respiratory rate or so-called therapeutic dehydration this minimum may need to be raised to between 4800 and 6000 cc per twenty-four hours in order to do away with the disabling signs and symptoms of toxic dehydration particularly in craniocerebral injuries. In spinal-cord and cauda-equina injuries this fluid intake serves chiefly to irrigate the genitourinary tract. It is the most efficient medicine that is available for the control of infection in that area, with its at

tendant aftermaths of renal and bladder stones encrusting cystitis pyelonephrosis renal abscess and functional deterioration of the bladder Secondly the fluid may serve to replace fluid lost in the serous discharge from large septic or granulating bedsores

Food and Vitamins

Except as noted under the heading *Bowels*, patients with central-nervous-system injuries may take any type of food by mouth if they are conscious and the food is palatable Unconscious or unco-operative patients must be fed through a tube. There need be no hesitation in using a tube under such circumstances It is best inserted into the stomach by way of one nostril and may be held in place by small strips of adhesive plaster attached to the face. All such adult patients and especially those with *spinal-cord injuries* must be given a minimum of 3000 calories a day The diet should also include enough protein to maintain a positive nitrogen balance This is not difficult in the case of craniocerebral injuries but is a real problem in the spinal-cord injuries. This is especially true in the presence of large bedsores anemia sepsis and similar conditions Experience has shown that at least 150 gm of protein per day must be *ingested* by all patients with spinal cord injuries and that more will be required if the patient has been allowed to become debilitated for any reason. It is virtually impossible to eat this amount of protein in any ordinary diet and have it palatable, so supplementary feedings, usually of milk or milk drinks re-enforced with some of the commercial amino acid preparations, must be added Occasionally vomiting and gastrointestinal upsets will accompany attempts to feed the patient such large amounts of protein. These arise from a failure on the part of the patient to metabolize the fat. They will usually disappear when the fat content of the diet or of the supplemental feedings is eliminated as far as possible. Above all, it is important for the doctor not only to know the detailed caloric and chemical constituents of the diet provided for such a patient but also to know that the food is actually ingested and kept down. A high vitamin intake is also essential in cases of spinal-cord injuries and is helpful in the others. Cevitamic acid, nicotinamide and yeast tablets or powdered yeast are minimum requirements and must be given in adequate doses and in many cases Betaxin, cod liver oil and riboflavin must also be added. Patients who must undergo a long convalescence should also be kept in the open air and sunlight as much as possible

Restraints

The problem of restraints chiefly affects the patients with craniocerebral injuries The need is closely linked to the headache and to the degree of coma that is present. The restlessness that comes from attempts to escape from the splitting, pounding headache of increased intracranial pressure or that is associated with abortive attempts to get to a urinal and thus avoid the necessity of micturating in bed will disappear with lowering of the intracranial pressure to normal and catheterization of the full bladder Failure

to recognize these needs entails the use of so much restraint as to be dangerous because of the associated immobility of the patient. At the worst, only that degree of restraint that will prevent the patient from doing harm to himself is all that is required. Adhesive plaster mitts that hold the fingers flexed around a roll of bandage in the palms a sheet across the chest and the attachment of the feet to the foot of the bed frame will prove ample in practically all cases. Often bed-sides are all that are needed. Spinal injuries rarely require restraint. The excitement and lack of co-operation seen in association with the anoxia of cervical-cord injuries are limited by paralysis. The important fact to remember in all cord cases is that immobilization with plaster of paris whether used as a means of restraint or as a splint, is contraindicated.

Drugs

The governing factor in the use of drugs, other than the chemotherapeutic and antibiotic groups, is the effect of the drug on the patient's respiration. Its effect on pain, excitement, stupor and so forth is secondary. Unless there are other overpowering reasons for their use, all drugs that are depressors of the respiratory centers are contraindicated in central-nervous-system injuries. This applies especially to morphine and the morphine derivatives. In particular, it may be taken as a general rule that morphine should never be given in either a craniocerebral or cervical-cord injury. The reasons are obvious, in that the former of itself jeopardizes the activity of the respiratory center and the use of morphine in the latter is characterized by anoxia and anoxemia because of the paralysis of all respiratory muscles except the diaphragm. As a substitute the barbiturates may be used. These will almost always be sufficient to control pain if in addition, steps are taken to empty the bladder extend and splint the neck and reduce or raise a higher or lower than-normal cerebrospinal-fluid pressure to a normal level. For convulsions that cannot be stopped by subcutaneous use of Luminal sodium, Pentothal sodium administered intravenously is the best therapy. Patients should be constantly watched during and after its administration however in order to avoid such complications as inhalation of vomitus and the like. Ether inhaled by the drop method, is the second choice. Paraldehyde given by rectum but not intravenously, is the most useful drug for the control of noisy excited patients. It is used in standard amounts.

Stimulants, the use of which is designed to combat general collapse in distinction to their use as specific antidotes for overdosage of depressants, rarely do much permanent good. Effects may be obtained by such drugs as aminophyllin and coramine but they are usually only temporary and probably have no more lasting effect than the camphor in-oil and ether that used to be given subcutaneously thirty-five years ago. The only stimulants that have any permanent effect are caffeine and strychnine. Even they are useless, unless given intravenously and in effective doses. They act as respiratory stimulants and as aids in combating peripheral vascular collapse. The dosage is $7\frac{1}{2}$ grains of caffeine and $\frac{1}{16}$ grain of strychnine given intra

venously alternately every one or two hours for six or eight doses. Other procedures, of course such as maintenance of a clear airway the use of whole-blood transfusions the application of external heat and the like are all equally essential. For hyperthermia of central origin it is best to use 30 grains of aspirin given by rectum every two to four hours, and tepid (not cold or alcohol) sponges. The patient is left moist and uncovered and evaporation with its cooling effect is enhanced by exposing him to a gentle breeze with the aid of an electric fan. Mineral oil and the ordinary bland cathartics supplemented by digital extraction the use of oil retention and milk and-molasses stimulating enemas will take care of all ordinary needs of the lower bowel. Urinary antiseptics *per se* are not needed as long as tidal drainage is used and provided that there are no stones abscesses, encrusted cystitis and the like present in the genitourinary tract.

Compartmentation of the Cranial Cavity and Mechanical Alteration of the Intracranial Pressure

Intracranial pressure may be mechanically altered by either decreasing or increasing the solid content of the dural cavity as well as by altering the amount of cerebrospinal fluid in the subarachnoid and ventricular spaces. The solid contents are compartmentalized within the various fossae of the skull and it has recently become apparent that one purpose at least, of the positive cerebrospinal-fluid pressure that is normally maintained in the central nervous system is to govern the movement of the various parts of the brain within these specific meningeal compartments. The corollary follows that increase in the contents of any one compartment with any shift that will interfere with intercompartmental movement of cerebrospinal fluid, will also modify the intracranial pressure. When this increase takes place in traumatic cases it is added to the alterations in the ventricular and subarachnoid contents capacity and shape that are caused by generalized endogenous cerebral edema, mechanical blockage of the absorption of the cerebrospinal fluid, the influence of cerebral venous pressure and the osmotic exchange of protein molecules in the capillary beds. Knowledge of these latter data and of their direct effect on the cerebrospinal fluid is widespread. The concept of increase in solid matter and its movement within compartments is new and less well known. Its importance appears to be equally great however.

If the cubic contents of the frontal compartment are increased the medial face of one cerebral hemisphere will be forced through the opening in the falx. If the contents of one temporal compartment are increased the medial surface of one temporal lobe will be forced into and through the incisura tentorii (Fig. 1). Both these changes—but especially the latter—cause interference with the flow of cerebrospinal fluid varying from a minimal slowing to the complete occlusion of the stream. The subarachnoid pathways on the surface of the brain stem are compressed and possibly done away with and the caliber and conformation of the aqueduct of Sylvius reduced and distorted. Increase in the cubic contents of the posterior compartment will

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either force the cerebellar tonsils downward through the foramen magnum or will force the upper surface of one or both cerebellar hemispheres upward through the incisura of the tentorium (Fig. 2) or both. In the first instance the fourth ventricular connection between the lateral ventricles and the cerebral subarachnoid spaces is closed in the second the same blockade of the cisternal subarachnoid current is produced as that caused by a temporal incisural herniation.

These processes, which are initiated as an increase in the solid contents of any one meningeal compartment and which terminate in the extrusion of



Fig. 1 Contusion and laceration of the brain with incisural herniation of a part of one temporal lobe. (Courtesy of Dr Raymond D Adams.)

a part of that content through a neighboring meningeal hiatus with eventual complete occlusion of the latter do not move directly from one extreme to the other. This is particularly true of the incisural herniations, and has been verified clinically. It is probably true in the foramen magnum herniations also but the clinical verification is not so clear. In the incisural herniations, the first step is a generalized shift of one cerebral hemisphere and the upper brain stem toward the side of the skull opposite to the overfilled compartment. As this progresses the opposite peduncle is pressed against the sharp edge of the opposing incisura. There is not necessarily any herniation of the temporal cortex as yet and there need not be and will not be any interference with the cerebrospinal fluid pathways, the flow of cerebrospinal fluid or the consequent universal leveling of the intracranial pressure until

herniation—as distinct from lateral displacement—has actually occurred. This is the condition and thus the shift in solid contents of the skull that produce the paradoxical ipsilateral hemiplegia that is frequently seen as a symptom of either an extradural or a subdural cerebral hematoma. Lumbar drainage during this stage will not produce a herniation. This comes only from further increase in cubic contents of one temporal fossa.

If the solid contents of one temporal fossa do continue to increase however and since a further lateral shift of the cerebral hemisphere and brain stem is no longer possible the temporal cortex immediately adjacent to the

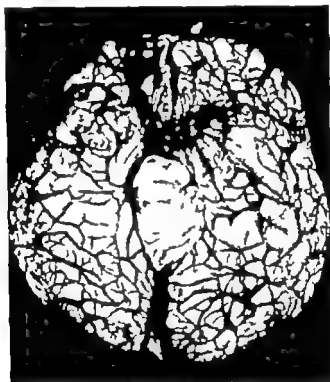


Fig. 2. Massive edema of the brain with herniation of the cerebellum upward through the incisura. (Courtesy of Dr. Raymond D. Adams.)

incisural hiatus is squeezed through this opening. This in contradistinction to the lateral shift of the hemisphere sets up a condition which is capable of being transmuted into a lethal compression of the brain stem, particularly if the resisting column of cerebrospinal fluid which is holding up the hernia's descent is removed or interfered with. Lumbar drainage in such circumstances is, at the very least, dangerous and will usually prove fatal unless steps are taken promptly to open the "bottleneck" and relieve the brain stem compression by enlarging the incisura or hernial opening. The same mechanical factors appear to govern herniation of the cerebellar tonsils through the foramen magnum.

Thus a pressure-counterpressure balance is set up with the tentorium as the fulcrum. The downward pressure exerted from above the tentorium by

hemisphere. Not only are such slitlike ventricles almost impossible to find, but also if found they contain so little cerebrospinal fluid that its removal will have virtually no effect on the intracranial pressure. Furthermore if the ventricle is large enough to contain a significant amount of cerebrospinal fluid and if this is removed, this very removal will permit a still greater increase in the bulk of the brain, which is of itself already enlarging because of the local trauma of the ventricular needle puncture. Drainage of the opposite ventricle by decreasing the resistance offered to the shift of the swollen hemisphere facilitates rather than prevents such herniation.

Mechanical Elevation of Pressure

Mechanical elevation of decreased intracranial pressure is necessary and may be lifesaving under certain circumstances. This was first shown by Gardner. Again the problem as pointed out above, is one of a shift in compartment contents, with resultant blockage of essential cerebrospinal-fluid streams. The condition is only seen in cranio-cerebral injuries after removal of large clots from the cerebral extradural or subdural spaces. The clots may be located in either the frontal or the temporal compartment, or in both. When the clots are removed the brain fails to expand to its normal shape and size and the lateral ventricle and subarachnoid space remain collapsed. It is useless to fill the space that has been recently emptied of clots with Ringer's solution. This only makes bad matters worse. In that the preoperative condition is reproduced. It is a matter of luck whether or not the ventricle can be tapped and forcibly expanded and there is also reason to suppose that any such sudden production of supratentorial increase in pressure may serve to change a reversible settling of the brain into an irreversible herniation. Attempted ventricular injection is therefore contraindicated. The procedure of choice is to introduce a needle into the lumbar subarachnoid space prior to closing the cranial wound and to inject through it, under pressure, enough warmed sterile Ringer's solution to lift the brain out of the compartmental foramina into which it has settled, thus re-establishing its normal contour and shape. This process usually must be, and may be, repeated as often as necessary during the following days, the criterion of need being a zero or near zero intracranial pressure as measured by lumbar puncture, and the criterion of amount of fluid to be used at any one such injection being the amount necessary to raise a subnormal intracranial pressure to a 150-mm. cerebrospinal-fluid pressure.

Respiratory Complications

Respiratory complications of injuries to the central nervous system are not to be regarded lightly. Their treatment merits an adequate diagnosis, made only by methods that are not of themselves harmful and that are used with care, with due consideration for their potential harm and with the proper tools. Nothing is easier to do—and nothing is potentially more harmful—than for the physician to say to the nurse "Leave the suction machine by the patient's bed and suck him out whenever he needs it." Respiratory

difficulties are caused in these patients for seven reasons. They are 1 The inhalation of a foreign body such as a tooth a piece of meat and the like 2 atelectasis from bronchial plugging 3 a tension pneumothorax 4 direct injury to the structures of the neck with apparent overactivity of the salivary glands 5 central paralysis of respiratory movements with the loss of the cough reflex 6 pulmonary emboli and 7 excessive intravenous administration of such fluids as normal salt solution. Each requires therapy in accordance with the fundamental cause.

Inhalation of a Foreign Body

This calls for prompt laryngoscopy and, if necessary, bronchoscopy with removal of the foreign body under direct vision. Repeated removal by suction, of the secretions stimulated by its presence will neither remove the foreign body nor alter the basic condition but will cause a complicating local traumatic edema of the tissues of the oropharynx and larynx. A tracheotomy can have no influence on the progress of this condition and should never be used.

Atelectasis from Bronchial Plugging

If thought of this complication can usually be easily diagnosed. Even in the absence of a definite diagnosis, and before resorting to suction, it is easier and less damaging to the patient to place him on his side with the suspected lung uppermost and his head low ask him to cough if he is conscious and at the same time strike the upper side of his chest vigorously with the operator's closed fist. If this does not dislodge the plug, bronchoscopy is indicated. Neither mechanical suction of the upper passages nor a tracheotomy can be expected to be of use in this condition.

Tension Pneumothorax

A tension pneumothorax with or without added blood in the chest cavity must always be suspected in any patient with a thoracic spine injury broken ribs a gunshot wound or a stab wound of the chest. It is an acute emergency in which proper therapy may be lifesaving. Thoracentesis with valve drainage of the pleural cavity can and should be done at once by any surgeon. The after-care and any necessary further therapy can then be left to the pulmonary specialist. Oropharyngeal, laryngeal and tracheal suction even with the aid of a tracheotomy is not only of no use but may actually be harmful.

Direct Injury to the Neck

Direct injury to the structures of the neck as a complication of either cranial or cervical-spine injuries may so interfere with swallowing that there appears to be excessive secretion in the respiratory tree. If the correct diagnosis is not made the mechanical suction designed to clear the oropharynx and larynx will further damage an already damaged structure. The difficulty

in swallowing caused by local pain will apparently necessitate feeding patient through an intubating stomach tube. The constant presence of such tube however causes further local harm and actually only makes matters worse. This is a problem for the laryngologist and should be referred to him at once.

If the basic injury is not recognized or has been neglected too long, the surgeon procrastinates, a tracheotomy may be necessary as a lifesaving measure in such cases and as the only way to circumvent the interference with respiratory exchange that is caused by the edema of the larynx or pharynx in such cases. Such need for a tracheotomy except in very severe injuries cannot be considered other than as the result of neglect, however.

Paralysis of Respiratory Movements

Paralysis of respiratory movements with apparent need for mechanical suction and possibly for tracheotomy may occur in connection with both cervical and dorsal spinal cord injuries as well as with cranio-cerebral injuries. Such paralysis caused by cord injuries should be easy to diagnose and can be effectively treated. This has been covered in detail in the section on cervical (see pages 56 and 67) and thoracic-cord injuries (see pages 56 and 67).

The relation to the trauma is less clear in the cranio-cerebral injuries. In these patients the later organic paralysis is first manifested as a functional paresis. The respiratory rate changes and may go either up or down. Excursion of the chest decreases, the rate becomes irregular and finally periodic and there is an apparent oversecretion of mucus in the respiratory tree. The patient's general condition deteriorates, he is more unconscious and less responsive, his cough reflex becomes less and less active and finally ceases to function and a diagnosis of "bronchopneumonia" is usually made. In the meantime mechanical suction done mechanically by the suction has been started and practiced more and more vigorously as the patient grows worse. Investigation will frequently reveal that suction and possibly intubating intratracheal tube were used during any preceding operation. Eventually it will become all too apparent that the patient is not only failing rapidly but that the secretions continue to collect in spite of virtually continuous suction. It is at this point that the doctor performs an "emergency tracheotomy." Thereafter more nurses are required for more suction through this new opening, thus thoroughly traumatizing the lower reaches, in addition to the already traumatized upper areas, of the respiratory tree.

Meanwhile the fundamental intracranial condition that is causing the respiratory difficulty continues to progress undiagnosed and untreated, to its predestined end. Neither the mechanical suction nor the tracheotomy is proper or indicated therapy for the fundamental etiologic condition. Removal of the respiratory secretions is merely symptomatic treatment and of no possible permanent therapeutic good. The cause lies in the damage to the brain. The resultant central respiratory deficiency is merely symptomatic evidence of this cause. The immediate cerebral pathology is edema

This increases locally at the site of the severest injury. In the continued absence of effective therapy the brain moves laterally across the skull compresses the brain stem against the opposite free edge of the incisura of the tentorium and finally interferes with the venous and cerebrospinal fluid circulations. If the condition is allowed to progress herniation of the temporal cortex begins through the incisura (see pages 7 and 38). The gradually increasing coma and severity of the patient's general condition lead to further inactivation of the cough reflex while at the same time paresis or paralysis of deglutition causes puddling and inspiration of the respiratory secretions. These latter are then changed from normal to abnormal and increased in amount by the trauma to the structures of the oropharynx and larynx caused by the constant and uncontrolled suction. If a tracheotomy is added the trauma is merely spread farther down the secretions further increased and bad matters made worse.

Such patients should be treated in accordance with their pathology and not in accordance with their most prominent symptom. They should be actively dehydrated and their intracranial pressure should be lowered by appropriate methods and any abnormal intracranial pathology such as clots, dealt with by an appropriate operation. If the patient continues to fail or if a "critical lumbar puncture" (see pages 7 and 19) is done the diagnosis of incisural herniation must be confirmed and, if present, treated by incision of the incisural ring. It is infinitely better to do any number of unnecessary cranial operations early than to postpone through such procrastination as a tracheotomy a curative operation till it is too late. As adjuncts postural drainage, minimal temporizing, suction under direct vision by a trained physician, avoidance of suction during operation and the avoidance of a tracheotomy without specific demonstrable indication for it are valuable and proper aids. If a high temperature is associated with the respiratory difficulty, tepid (body temperature) sponges and large doses of aspirin by rectum should be added (see page 22).

Never under any circumstances should the responsibility for the mechanical suctioning of or the doing of a tracheotomy in these patients be foisted on the intern, the resident or the nurse by the responsible surgeon.

Pulmonary Emboli

Pulmonary emboli for some reason are extremely infrequent complications of trauma to the central nervous system. The treatment of the site of origin and the best methods to be used in the prevention of further emboli should be decided on after consultation with the vascular specialist and in the light of the patient's over-all picture. It has been recently demonstrated by Faxon and his co-workers¹ that part of the lethal effect of pulmonary emboli can be attributed to reflex radial bronchiolar spasm. They have shown further that this spasm can be released and when released is followed by dramatic and often lifesaving return of the respiratory exchange to nearly normal. This is brought about by the injection through the neck, of the inferior cervical or stellate ganglion. The earlier this is done after

the arrest of the embolus in the lung, the better the effect. It may be tried, however as late as three weeks after the lodgement, with a fair expectation of less but definite improvement. Only one side (that of the infarcted lung) should be injected, and the injection may be repeated. Even if this is of no effect, it does no harm. Neither mechanical suction nor tracheotomy is of any use in the treatment of pulmonary emboli.

Excessive Intravenous Injection

Excessive intravenous injection of such isotonic solutions as normal saline can produce an escape of fluid into the lungs that may cause the patient to drown in his own secretions. The critical amount will vary with the patient's general, pulmonary and cerebral conditions as well as with his blood pressure. Excessive fluid intake is commonly associated with an injudicious attempt to treat surgical shock by the administration of normal salt solution intravenously. Any benefit, in terms of increased blood or pulse pressure is purely apparent and at most transient. If the administration is continued, the osmotic relationship between the capillary beds and the tissue fluids is so altered as to encourage escape of electrolytes in solution into the tissues. Thus, dependent and peripheral edema is succeeded by pulmonary edema. The process is, for practical purposes, self-perpetuating and is particularly likely to occur in patients with severe cranio-cerebral injuries. Attempts to raise peripheral blood pressure should be limited to the use of 50 per cent glucose solution (as a temporary measure) plasma in the absence of whole blood and, by preference whole blood given intravenously. Amounts of fluids given for other purposes (such as hydration) must be controlled by a careful daily graphing of the fluid intake and urinary output. Continued approximation and, even more importantly crossing of the curve of fluid output to a point higher than that of the curve of intake is a danger sign that must not be disregarded.

Suction and Tracheotomy

It cannot be too strongly emphasized that, with certain specific exceptions the respiratory complications of trauma to the central nervous system will respond better without oropharyngeal and laryngotracheal suction. If this must be done at all the suction must be carried out by a member of the hospital staff who is above the resident level who is not a nurse and who manipulates the suction tip only under direct vision. Suction should be regarded as a major surgical procedure. It should never be attempted unless a proper tube that has been properly coiled and refrigerated is used. This tube should not be inserted into the respiratory tree without the use of a laryngoscope. The operator must be cognizant of the anatomy and physiology of the area in which he is working. Tracheotomy is, with rare exceptions, no more than an excuse for inadequate diagnosis. Justification for its use must be specific, precise and based on sound knowledge of the pathology involved. Its routine use or use on less accurate grounds cannot be condemned too strongly.

Anesthesia

In general the best anesthesia to use in treating injuries of the central nervous system is the one that best fits the individual case. It may be taken as an unbreakable rule however that whatever general anesthetic is used local anesthesia with 1 or 2 per cent procaine combined with adrenalin and used in the soft tissues and in appropriate nerve roots is essential and must always be included.

Within these wide limits however my experience has satisfied me that certain anesthetics and certain procedures are better avoided if at all possible. Among these are nitrous oxide badly given drop ether and Pentothal sodium that is administered too rapidly. No matter how well given or whether or not given as a preliminary to some other anesthetic practically speaking nitrous oxide is an asphyxial agent as far as the central nervous system is concerned. Congestion followed by edema and excessive unnecessary bleeding during operations on and around the brain and spinal cord is practically inevitable. Patient morbidity and possibly also the mortality is thereby increased.

Ether if properly given by the drop method on an open gauze cone, is still the safest general anesthetic for this group of patients, and especially for children. Improperly given with resultant anoxia, cyanosis and laryngeal spasms or preceded by nitrous oxide, it can be the worst and most dangerous of anesthetics. It is true that it raises the intracranial pressure, but thus, in the absence of anoxia and vascular congestion is of little moment as compared with its safety. Care must be taken to prevent explosions if electrical equipment is in use however. Ether is a heavy gas and tends to sink to the lower air levels or the floor of the room.

Unless contraindicated by age or the presence of hepatic or renal disease, Pentothal sodium, given intravenously and supplemented by local anesthesia, is the most satisfactory of all anesthetics and is almost as safe as ether but cannot be used in children. Certain precautions must be taken. It must not be allowed to enter the perivascular spaces. It should not be given as a constant drip. It must be injected very slowly or else laryngeal spasm will be caused and it may be necessary to give oxygen with it. These three anesthetics—local ether and Pentothal sodium—will fulfill all anesthetic needs in patients with injuries to the central nervous system except in the case of infants, who do best with local anesthesia supplemented by whiskey. To give the latter the baby is allowed to suck a liquor impregnated piece of gauze.

Among the dangerous procedures are the use of intratracheal tubes and "blind" or "routine" suction. Intratracheal tubes are contraindicated in all patients with central nervous-system injuries. Those who need them most, that is those with cervical-spine injuries, are the patients in whom it is most dangerous to attempt their insertion. Mechanical suction of the oropharynx, and particularly of the larynx and trachea should be studiously avoided, except under direct vision and for definite specific indications. Positioning of the patient's head on the table avoidance of laryngeal spasm, the use of

a properly fitting mouth-airway and the choice of a suitable anesthetic properly administered will obviate the need for suction in almost all instances "Routine suction" is particularly pernicious.

THE TREATMENT OF CRANIOCEREBRAL INJURIES

The Nonoperable Group

All these patients must be hospitalized for therapy Table 1 summarizes the pathology condition of the cerebrospinal fluid and intracranial pressure, means of diagnosis, symptoms, signs and treatment of these injuries.

Concussion of the Brain

The *diagnosis* is made on the basis of a history of unconsciousness immediately following a blow on the head and associated with preaccidental amnesia either alone or in combination with accidental amnesia absence of *symptoms* with recovery of consciousness a normal physical and neurologic examination normal cerebrospinal fluid and normal intracranial pressure Postaccidental amnesia also occurs but its value in diagnosis has been largely destroyed by the fact that patients who either have or claim to have had a craniocerebral injury and who claim postaccidental amnesia believe it to be an essential symptom in establishing their claim to an injury and know that it may be difficult or impossible to verify the accuracy of their statement. On the other hand lay knowledge of the other types of amnesia is virtually nonexistent. The latter therefore are much more reliable and hence valuable from a diagnostic point of view Furthermore the accuracy of the statements made by patients concerning preaccidental and accidental amnesia can be ascertained without too much difficulty The *pathology* is not known. No *treatment* is necessary

Edema and Congestion of the Brain

This is commonly associated with *concussion* the exception being injury caused by slow crushing or a stab or bullet wound of the skull and brain, the latter being caused by missiles of small caliber In these circumstances *concussion* is not present. Otherwise the *diagnosis* is made on the basis of the history as given under *Concussion* and by the cerebrospinal fluid and intracranial pressure findings. In addition, there is the presence of preaccidental accidental and postaccidental amnesia, unconsciousness postinjury headaches dizziness and "nervousness." There may be a multiplicity of reflex changes, and possible hemiplegia and convulsions, especially in children. The cerebrospinal fluid is under measurable increased intracranial pressure for a variable length of time but is otherwise normal The *pathology* includes that of *concussion* and is as follows an increase in brain volume with a reduction in the size of the cerebrospinal-fluid reservoirs and an increase in the intracranial venous pressure resulting from a slowing of the absorption of the cerebrospinal fluid from the subarachnoid space. The *treatment* may be either reduction of the brain volume or mechanical with drawal of the excess unabsorbed cerebrospinal fluid as follows

1 REDUCTION OF BRAIN VOLUME. A Administer intravenously 200 cc of a 50 per cent solution of sterile dextrose in normal salt solution or from commercial sealed ampoules every two hours for three doses. This may be repeated after an interval of twelve hours and again after another twenty four hours if necessary. Care must be taken to keep the solution out of the perivenous tissues. To avoid clotting of the vein after the glucose has been given it should be washed out at once with 25 cc. of sterile isotonic salt solution.

B Administer 2 ounces of saturated solution of magnesium sulfate by rectum every three hours for four doses or every four hours for three doses according to the amount of edema and the effect desired. This can be repeated after a twelve-hour interval and again after another twelve hours. The solution should be warmed to body temperature and must be given by gravity through a No. 10 French soft rubber catheter which should be left in the rectum and clamped off so that it will not leak. If these precautions are not observed, either the rectum will reject the solution or the latter will come out with the catheter when it is removed. To be effective the solution must remain in the rectum for at least thirty minutes.

2 MECHANICAL WITHDRAWAL OF EXCESS UNABSORBED CEREBROSPINAL FLUID. This is accomplished by lumbar drainage of the subarachnoid space. A lumbar puncture is performed painlessly (page 235). The initial intracranial pressure is measured on a manometer and if it is above 150 mm. of water and the patient is relaxed and over ten years of age enough cerebrospinal fluid is withdrawn to reduce it to that level. If the level is 150 mm. of water or lower no fluid is withdrawn unless the puncture is the first made since the accident. In this case $\frac{1}{2}$ cc. is withdrawn to determine grossly and by cell count whether or not blood is present. If the patient is ten years of age or younger the critical level of cerebrospinal-fluid pressure is 100 mm. of water rather than 150 mm. Under no circumstances should compression of the jugular veins be carried out, because this increases an already dangerous cerebro-venous congestion and raises still higher an already pathologically increased cerebral venous pressure. Such lumbar drainage should be performed once every twenty four hours until two successive normal pressures are recorded prior to the removal of any cerebrospinal fluid. If three days later another lumbar puncture still shows normal pressure no further punctures are necessary.

3 THE CRITICAL LUMBAR PUNCTURE. *General Considerations* Under certain conditions having to do with intracranial compartmentation and local increase of the contents of any one compartment (see page 7) the therapeutic lumbar drainage described above may prove to be the one factor that might cause herniation of the temporal cortex through the incisura tentorii or herniation of the cerebellar tonsils through the foramen magnum. In the first instance the condition is reversible if recognized and treated in time. In the death or decerebration from compression of the brain stem being otherwise inevitable. Suspicion on the part of the surgeon that such a condition may be developing or may actually exist should be promptly aroused on the

TABLE 1 NONOPERABLE CRANIOCEREBRAL INJURIES

DIAGNOSIS	PATHOLOGY	CEREBRO-SPINAL FLUID	INTRACRANIAL PRESSURE	DIAGNOSIS MADE BY	SYMPTOMS	SIGNS	TREATMENT
Concussion	Unknown	Normal	Normal	History of unconsciousness and amnesia	Unconsciousness only	Preaccidental and/or accidental amnesia	None
Edema and Congestion	Increase in brain volume, reduction in size of C.S.F. reservoir, increased intracranial venous pressure	Normal	Increased ++	History as above and lumbar puncture findings	Unconsciousness, headaches	Preaccidental and/or accidental amnesia, reflex changes, hemiplegia and convulsions in children	Either reduction in brain volume by dehydration therapy or removal of excess C.S.F. by lumbar drainage from the shrunken reservoir
Continued and/or exacerbation	Increase in brain volume, reduction in size of C.S.F. reservoir, increased intracranial venous pressure, interference with the rate of absorption of C.S.F. into the blood cells in C.S.F. with mechanical occlusion of arachnoid, with and mechanical blockade of C.S.F. absorption	Bloody varying from light pink to virtually pure blood	Increased +++	History as above and lumbar puncture findings	Unconsciousness, headaches, confusion, excitement, coma, convulsive seizures, change in personality etc.	Preaccidental and/or accidental amnesia, reflex changes, peripheral and central paralysis, convulsions, hemiplegia, decrease in brain rigidity, sensory changes, cortical and subcortical atrophy etc.	Removal of excess C.S.F. by mechanical means from the shrunken reservoirs and the mechanically blocked subarachnoid space, i.e., by lumbar drainage. This may be supplemented by but not replaced by, reduction of brain volume by dehydration therapy
Toxic dehydration	Abnormal decrease in brain volume, increase in size of C.S.F. reservoir, acidosis in children	Normal or bloody	Decreased below the level appropriate for the cerebral damage	History as above and lumbar puncture and fluid balance findings	Fever, confusion, drowsiness, reduction of earlier symptoms that have disappeared	Rise in temperature, abnormal CO ₂ , combining power, acidosis, retardation of earlier signs of brain damage, altered fluid intake and output relationship	Fluids, preferably in the form of water and preferably by mouth up to at least 5000 and possibly 6000 cc. per 24 hours for a normal adult until fluid intake and output relationship and intracranial pressure are again normal
Bone injury	Any type of noncompounded or nondepressed fracture	No influence on the C.S.F.	Not affected by the bone injury	X-ray	None	None	None

appearance of certain phenomena in connection with any lumbar puncture that is accompanied by cerebrospinal fluid removal. Such a procedure is described as a "critical lumbar puncture." The demonstration of a critical lumbar puncture may necessitate an emergency operation (see pages 7 and 224) which if done promptly will save life and restore normal function but which if done less promptly will only save life at the expense of decerebration and may not even do that. The crucial period is six hours after the performance of the critical lumbar puncture.

The Criteria. The critical symptoms are as follows:

1. An intracranial pressure of 300 mm. or more of cerebrospinal fluid in a quiet patient.
2. The sudden complete cessation of the flow of cerebrospinal fluid during lumbar drainage.
3. The reduction by lumbar drainage of any high cerebrospinal pressure to 0 mm. of cerebrospinal fluid.
4. An immediate progressive increase in coma or the development of respiratory irregularity, periodicity or inadequacy or both immediately after the puncture is finished.
5. A progressive steady change for the worse in the patient's general condition immediately after the puncture.

Any one or any number of these findings, in any combination at once raise the suspicion of the herniation of the temporal cortex through the incisura or of herniation of the cerebellar tonsils through the foramen magnum. If in addition the patient is suspected of or is known to be or to have been, suffering from a temporal subdural or extradural hemorrhage, a severe temporal or temporofrontal contusion or laceration with local swelling or a temporal intracortical hemorrhage or a hemiparesis or hemiplegia proven to be ipsilateral to a cerebral subdural or extradural hematoma, the likelihood of an incisural herniation of the temporal cortex is virtually so great as to necessitate immediate surgical visualization and verification.

4. GENERAL THERAPEUTIC MEASURES. The following general therapeutic measures are applicable to all cases, whether treated by dehydration or lumbar drainage. *The intracranial pressure* should be checked by actual measurement on a manometer at each lumbar puncture. *Fluid intake* should be maintained at 4500 cc. per twenty-four hours, unless the patient is under ten or over sixty-five years of age and if the renal function is normal and there is no edema of the lungs, ankles or dependent soft tissues. Care should be taken to prevent administration of an excess of salt when the fluid is given intravenously or when large amounts of whole blood have been given by transfusion. *The potassium balance* should be kept at normal levels. The diet should contain 3000 calories and 80 to 100 gm. of protein and the patient's nitrogen balance should be maintained. Otherwise the diet should be as nearly normal as the patient will eat. *The urinary bladder* should not be allowed to go unemptied for more than eight hours. Retention should be dealt with by catheterization and if necessary by the use of tidal drainage (see page 84). Overflow or other incontinence should be

cared for by tidal drainage. *The bowels* should be controlled with soap suds, oil or milk-and molasses enemas as necessary but only until mild catharsis with cascara, Phenolax, milk of magnesium or the like may be substituted if needed to produce normal evacuation. *Restlessness* should be treated with 5 grains of Luminal sodium subcutaneously every four hours if necessary or by $\frac{3}{4}$ grain of phenobarbital by mouth three times a day. The need for this drug therapy usually will be obviated if the bladder is emptied, the intracranial pressure kept at normal and restraints reduced to a minimum. *Morphine* and its derivatives are contraindicated. *Restraints* must be minimal: bed sides, a sheet fastened over the chest to the bed frame, attachment of the patient's feet to the foot of the bed, quiet and minimal lighting and especially no direct or ceiling light. Adhesive plaster mitts, relief of headache by reduction of intracranial pressure and maintenance of an empty bladder are usually effective and all that is necessary. Restraints are intended only to prevent the patient from harming himself and are not to be used to immobilize him or his nurse. *Excitement*: Three ounces of paraldehyde by rectum every three or four hours should be used as indicated. *Coma*. As long as the patient is in a coma he must be moved every two hours and so placed at all times that his airway remains clear and secretion or vomitus may drain from his mouth and cannot be inhaled. A pillow under the head is usually best, but the condition of the airway will determine this.

Mechanical suction of the nasopharynx and oropharynx should not be practiced except for specific indications and never by a nurse. Tracheal intubation with suction of the larynx under direct vision, if done by a specialist, may be resorted to if airway obstruction is present at these levels. Pulmonary atelectasis, if not relieved by such procedures as positioning and induction of coughing, should be treated by bronchoscopy and removal of the obstructing plug under direct vision. *Tracheotomy* is contraindicated. All the above procedures designed to keep the patient's airway clear are potentially as harmful as they are helpful. They should be used only by one skilled in laryngoscopy and never by a nurse or intern. This applies as strongly to pharyngeal suction as it does to laryngeal or intratracheal suction (pages 13 and 16). *Arms and hands* must be kept off the chest and abdomen and not allowed to get beneath the body. *Maintenance of the intracranial pressure* at normal is the only effective direct therapy for coma.

Emergencies. **SURGICAL SHOCK**. Whole-blood transfusions must be given in adequate amounts and repeated as often as necessary to restore the pulse pressure to normal.

PERIPHERAL VASCULAR COLLAPSE. Repeated small (200 cc.) blood transfusions $\frac{1}{15}$ grain of strychnine intravenously every two hours for four doses, alternating with $7\frac{1}{2}$ grains (1 ampoule) of caffeine intravenously every two hours for four doses, is the most effective therapy.

ACUTE CENTRAL RESPIRATORY FAILURE. Strychnine and caffeine given as described (above and page 6).

HYPERTHERMIA OF CENTRAL ORIGIN. Thirty grains of aspirin by rectum

every three hours and tepid (normal body temperature) sponging of the body and extremities followed by their exposure while wet to a gentle breeze from an electric fan should be used whenever the temperature reaches 103° F or over. Alcohol and cold water rubs and cold packs are contraindicated. Acute central respiratory failure and hyperthermia of central origin may develop because of compression of the brain stem by a temporal lobe herniation through the incisura of the tentorium cerebelli or because of compression of the hypothalamic region by herniation of the medial aspect of a cerebral hemisphere through the opening beneath the falx. In either case the only effective therapy is surgical, and the medicinal measures mentioned are of temporary effect only. These conditions and their treatment are described under *Herniation through the Incisura* (pages 38 and 224).

Convulsions. Pentothal sodium, given intravenously and very slowly until the patient is anesthetized is the first therapeutic choice. This may be repeated once or twice if necessary. If more anticonvulsant is needed as an emergency measure either given by the drop method and inhaled through a gauze mask is preferable. After the convulsions have stopped, $\frac{3}{4}$ grain of phenobarbital three times a day or $1\frac{1}{2}$ grains of Dilantin three times a day should be started and continued until the cause of the convulsions can be diagnosed and corrected.

Position. Ordinarily a pillow should be placed under the head other wise, that position which ensures an open airway and that is most comfortable for the patient is the best one.

Sleep. Drugs that induce sleep should not be used if it is at all possible to avoid them. One of the barbiturate group is preferable if any must be used. When patients are convalescent, they should spend eight hours in bed but may not (and will not need to) sleep that long until they have started to exercise. With exercise will come a compensatory increase in sleep. Associated "nervousness" will usually disappear with reassurance by the doctor that less than eight hours sleep in the absence of exercise is harmless.

Length of Stay in Bed. The patient should be kept in bed until such time as he is known to have had a normal intracranial pressure (neither too high nor too low) for two successive weeks.

Length of Hospital Convalescence. The patient should stay in the hospital until he is able to remain out of bed all day and to walk up and down two flights of stairs in succession without aid. These requirements must be accomplished on the patient's own initiative.

Home Convalescence. For the first two weeks after leaving the hospital the patient should have one hour's rest before lunch and supper otherwise the only limitations on his activities are those imposed by his physical capacity. The rest periods are stopped after the first two weeks. Thereafter gradually increasing regular morning and afternoon exercise in the form of measured walks of such length as to provide exercise without either producing exhaustion or encouraging laziness should be prescribed. The length of the walk is to be gradually increased, according to the patient's decision until he is walking five miles in the morning and five miles in

the afternoon. It is important that this exercise be carried out regularly every morning and every afternoon. At this time the patient is physically—and should be psychologically—ready for discharge to his preaccidental activities. The only future limitations that should exist will be those caused by permanent destructive lesions of the brain. Patients with convulsive seizures should be continued on anticonvulsant therapy (see *Incisural Herniation* page 38)

Contusion and/or Laceration of the Brain

This is commonly associated with *concussion* and always with *edema and congestion* of the brain. The diagnosis is made according to the history as given under *Concussion* (page 18) and on the basis of the cerebrospinal fluid and intracranial pressure findings. The signs and symptoms are those of concussion and edema in an aggravated form. Unconsciousness is deeper and more prolonged. Lack of co-operation extending to mania, peripheral and cranial nerve paralyses, surgical shock, respiratory, circulatory and thermoregulatory abnormalities and blood in the cerebrospinal fluid, varying from only enough to faintly stain it to so much as to give it the appearance of pure blood, are common findings. Unless the patient is in surgical shock and is dehydrated, the intracranial pressure is high and may rise to 400 to 500 mm. of water (page 10). The pathology includes that of concussion and edema and congestion and is as follows. There is a bruise (contusion) or tear (laceration) of at least the surface of the brain (see Fig. 1 page 8) with extravasation of free red-blood cells into the subarachnoid space and resultant plugging of the arachnoidal villi and mechanical blockage of the absorption of the cerebrospinal fluid into the large cerebral venous channels and backing-up and distention of the subarachnoid and ventricular spaces. Treatment must be by mechanical removal of the excess mechanically blocked, unabsorbed cerebrospinal fluid. This will lower the intracranial and cerebral venous pressures, reduce cerebral congestion and brain volume and improve the cerebral circulation and oxidation. Removal of the blood contained in the subarachnoid or other spaces is an incidental accompaniment of this procedure and is not one of the objectives. Dehydration therapy can be used to supplement this mechanical drainage but cannot replace it and is not efficient when used alone. It should be carried out as follows:

1. Dehydration therapy should be carried out as described under *Treatment Section 1—Edema and Congestion* (page 19). In patients with contusion and laceration of the brain this method is most useful as an emergency measure; hence the intravenous route is preferable. Care should be taken not to carry its use to a point where toxic dehydration (see below) is produced, in the belief that because a little dehydration is good more is better.

2. Mechanical drainage is carried out as described under *Treatment Sections 2 and 3—Edema and Congestion* (page 19) except that in the most severely injured lacerated brain cases the drainage by lumbar puncture

ture may have to be repeated two three or four times during every twenty four-hour period as long as the cerebrospinal fluid pressure remains at very high levels (350 to 500 mm. of water) and contains enough blood to give it the appearance of virtually pure blood, and unless the lumbar puncture gives evidence of being a *critical lumbar puncture* (page 19)

3 The general measures of therapy detailed under *Treatment Section 4—Edema and Congestion* (see page 21) apply here and should be used in the treatment of contusion and laceration of the brain (see also *Incural Herniation* page 38 and *Critical Lumbar Puncture* page 19)

Toxic Dehydration with or without Acidosis

The diagnosis is made by the history as given under *Concussion* and *Edema and Congestion* and by a study of the cerebrospinal fluid and intracranial pressure findings and of the fluid intake and urinary output relations. Acetone and diacetic acid may or may not be found in the urine and there is usually a reduction in the carbon dioxide combining power of the blood. Toxic dehydration may be associated with any form of cranio-cerebral injury including concussion. In addition to the *signs and symptoms* characteristic of the associated brain injury there will be a tendency for such of these signs and symptoms as have already disappeared to recur without obvious reason unconsciousness that has been decreasing will again increase reflex changes and cranial nerve palsies that have been disappearing will reappear or increase rather than decrease the patient will become irritable his temperature will rise and his skin will become dry. In severe cases the tongue will be dry and coated and the eyeballs sunken. The cerebrospinal fluid pressure will be lower than it should be on the basis of the characteristics of the fluid, the associated brain injury the length of time since the accident and the treatment that has been given. The twenty four hour urinary excretion will be below the level appropriate for the twenty four hour fluid intake. At operation the subarachnoid space will be on the dry side and the brain noticeably shrunken. The *pathology* is that peculiar to an abnormal relation between fluid intake and output. The *treatment* is to administer enough fluid to restore this relation to normal and to stop all therapy that has a dehydrating effect. Fluid should be given (preferably by mouth) at the rate of 6000 cc. per twenty-four hours for a normal adult with normal circulation and kidneys. This amount should be scaled down for cardiac and renal invalids and children. Glucose in the form of large amounts (1 pint three times a day) of fresh orange juice and sugar on all appropriate foods should be given particularly in the case of children. If fluids and food cannot be taken by mouth a tube should be placed in the stomach through the nose and the feedings given through the tube. If this is not possible, the fluids should be given by vein in the form of a 10 per cent solution of glucose dissolved alternately in isotonic salt solution and sterile distilled water. Accurate graphs of the fluid intake and urinary output should be kept and the above treatment continued until the urinary output curve is parallel to and approximates the fluid intake curve. A daily meas-

urement of the cerebrospinal-fluid pressure should be made on a manometer so that the increase in intracranial pressure that frequently appears with proper dehydration can be dealt with at once by mechanical lumbar drainage of the backed-up unabsorbed cerebrospinal fluid and the intracranial pressure be thus reduced and maintained at normal levels (see page 19). All other necessary therapy is that which is appropriate to whatever cranio-cerebral injury happens to be present. It should not be neglected because of the associated toxic dehydration any more than the latter should be neglected because of the associated brain injury.

The Bone Injury

In this nonoperable group of craniocerebral injuries the bone injury is limited by definition to that variety that is neither compounded (whether externally or internally into a nasal sinus or the ear) nor depressed. Such fractures are diagnosed by x ray and produce of themselves no symptoms, either at the time of the injury or later. They therefore require *no treatment*.

The Operable Group

All patients must be hospitalized for therapy

Meningeal Hemorrhages

Table 2 summarizes the pathology condition of the cerebrospinal fluid and intracranial pressure, diagnosis symptoms signs and treatment. (See also *Incurial Herniation* page 38.)

EXTRADURAL HEMORRHAGE (Fig. 3) The *diagnosis* is made by a suggestive history (the so-called pathognomonic history occurs in only 40 per cent of cases) with possible confirmation by x-ray demonstration of a fracture line that crosses either the middle meningeal vessels or one of the large venous sinuses. Such findings of themselves make an exploratory diagnostic trephination obligatory. The final diagnosis is made after operative visualization of one or both of the cerebral or cerebellar extradural spaces. The *signs and symptoms* are those of the associated brain injury (which is always present) in addition to those of rapid compression of the brain (in the patients with clots of venous origin the compression is less rapid) the signs varying according to the area of the cortex being subjected to the greatest pressure. In certain instances and probably when the increase of the solid contents of the involved temporal fossa starts a shift of the brain toward the opposite side of the skull (see page 38) the signs may be ipsilateral to the clot rather than bearing the classical contralateral relationship. The cerebrospinal fluid will have the characteristics appropriate to the associated brain injury. The intracranial pressure will usually be higher than normal, but not necessarily so. The *pathology* which includes that of any associated brain injury is as follows. There is a rapidly expanding clot developing at any point in the extradural space, with either an open or closed tear in the trunk or in one of the branches of the middle meningeal artery or vein or of one of the large venous sinuses or one of their larger tributaries. *Treatment* of the extradural

hemorrhage is operative and that of the associated brain injury is that indicated above under the appropriate heading. These patients should be hospitalized for treatment in a large well staffed properly equipped hospital that has a blood bank or its equivalent available. They can and should be moved for any reasonable distance by train plane or ambulance to conform with this requirement, because there is less risk from such transportation than there is in attempting to operate on such a patient in a small

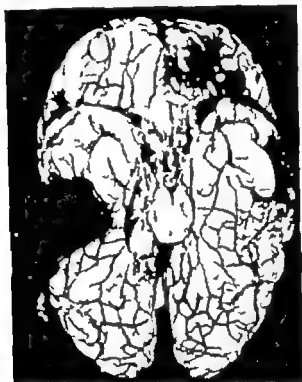


Fig. 3 Extradural temporal hematoma with ipsilateral incisural herniation of the temporal cortex. (Courtesy of Dr. Raymond E. Adams.)

poorly equipped hospital that has no means of providing large amounts of blood for transfusion and whose staff is inadequate in number and improperly trained.

The operation should not be of a bone-flap type. It should be transtemporal, with the opening in the bone and dura enlarged to include all the squama and as much more bone posteriorly medially or anteriorly as is necessary to permit adequate evacuation of the clot as well as visualization and closure of the ruptured vessel. After removal of the hematoma the dura should be left open to provide a decompression. Whole blood should be administered by transfusion before, during and after the procedure. If the clot is cerebellar the approach should be by the suboccipital route and the dura should not be left open when the wound is closed. In either case the subdural space may be drained for thirty-six hours if necessary. (See also *Incisural Herniation* page 38.)

TABLE 2. OPERABLE CRANIOCEREBRAL INJURIES
Meningeal Hemorrhages

DIAGNOSIS	PATHOLOGY	CEREBRO-SPINAL FLUID	INTRACRANIAL PRESSURE	DIAGNOSIS MADE BY
Extradural hemorrhage	Rapidly expanding clot caused by the rupture of either the middle meningeal artery or a venous sinus, always associated with some degree of brain injury and almost always with a fracture	Normal or bloody	Usually but not necessarily increased	A suggestive history with possible confirmation by x-ray. This much makes an exploratory trephination (at least) mandatory. Final diagnosis made at operation.
Subdural hemorrhage	Slowly expanding clot that may be pure blood (solid type) or mixture of blood with C.S.F. (mixed or fluid type), or a collection of high protein fluid enclosed in a fibrous membrane (encysted type, hygroma, hygroma) some degree of brain injury but not necessarily a fracture; rare instances of clots from a ruptured bridging vein without brain injury; clots commonly bilateral and prone to recur postoperatively	Normal or bloody	Usually but not necessarily increased	Bilateral exploratory trephination, usually temporal. Failure of the patient to improve or an increase in signs or symptoms makes operative exploration mandatory

Intracranial Hemiation of Temporal Cortex

Herniation of uncus and adjoining temporal cortex through foramen, with brain stem compression	Shift of involved hemisphere and brain stem toward opposite side of skull; compression of contralateral peduncle on edge of incisura; squeezing of temporal cortex into incisural hiatus with potential herniation and partial block of cerebrospinal fluid flow; herniation of cortex through the incisura with increasing block of cerebrospinal fluid flow; supratentorial edema with increase in C.S.F. pressure, thrombosis, ischemia, softening and liquefaction of temporal cortex, compression of brain stem and structures on its surface including cranial nerves 3, 4 and 6 and posterior cerebral artery; edema, thrombosis and hemorrhage of brain stem, distortion and partial occlusion of aqueduct of Sylvius; herniation of supratentorial cortex through hiatus in falx, degenerative decerebration	Normal or bloody	May be high, low or normal; may suddenly drop to 0 with or without cessation of flow of C.S.F. during lumbar puncture may be measured lower than clinical evidence suggests it should be	A history of head injury with failure of the patient to improve with usual therapy for the pathology as diagnosed other than herniation. Patient has increasing coma, respiratory abnormality and usually have a meningeal hemorrhage with or without a lacerated brain, often has a contusion or hemiparesis or hemiplegia ipsilateral to the clot and a "critical lumbar puncture" (see p. 19) may have been done. The tentative diagnosis of herniation is confirmed by the demonstration of cerebrospinal fluid trapped under pressure beneath the tentorium with resistant spurring through a stab wound in the tentorial leaf or the welling up of large amounts by way of the nucleus after retraction of the herniated temporal cortex.
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TABLE 2. (Continued)
Meningeal Hemorrhages

SYMPTOMS	SIGNS	TREATMENT
Those of the associated brain injury. These cause the first period of unconsciousness.	Those of the associated brain injury. There may be a contralateral, ipsilateral or no hemiplegia with or without aphasia. A dilated pupil may be either ipsi- or contralateral to the clot.	Emergency operation with removal of the clot; mechanical closure of the bleeding vessel and subtemporal decompression in addition to that treatment needed for brain injury.
Those of rapid compression of the brain by the growing clot. These cause the second slowly developing period of unconsciousness.		
If the first unconscious period overlaps the second there is no conscious interval and hence no classical so-called pathognomonic history. If it does not overlap there is a conscious interval and hence a "pathognomonic" history.		
Those of the associated brain injury of increased intracranial pressure and of post-traumatic seizures. There are no characteristic or pathognomonic symptoms of a subdural hemorrhage.	Those of the associated brain injury, and of increased intracranial pressure, local circulatory distortion, occlusion of the cerebral subarachnoid space. There are no characteristic or pathognomonic signs of a subdural hemorrhage.	Removal by operation of the hematoma including its membranes, together with drainage of the subdural space, usually through and with a subtemporal decompression on one side at least. The procedure should be bilateral.

Incidental Herniation of Temporal Cortex

Those of the associated brain injury: increasing coma, respiratory abnormality, hemiparesis and hemiplegia, convulsive seizures, decerebration, a "critical lumbar puncture" (see p. 19).	Increasing coma, respiratory abnormality, dilated fixed pupil or pupils, stiff neck, Magnus-deKleyn reflexes, vomiting, irregularity of pulse, blindness in one half the visual field, unilateral or bilateral Babinski reflexes, fever, a "critical lumbar puncture" (see p. 19), the signs of any associated brain injury.	<i>Precautions.</i> Routine division of one tentorial leaf and its incisural edge whenever a meningeal hemorrhage is demonstrated. The same procedure whenever a patient goes downhill in spite of other wise adequate therapy; whenever a "critical lumbar puncture" is performed (see p. 19) and within 6 hours of its performance. Postpyramidal exposure of tentorium, stab wound of avascular portion of leaf, avoidance of injury to the bridging veins, incision of tentorial leaf through the incisural ring with folding back of the newly created tentorial flaps, reduction, if necessary of the temporal cortical herniation.
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SUBDURAL HEMORRHAGE *Acute* The diagnosis is made by exploratory operation and is suspected when a patient whose condition has been diagnosed as belonging to one of the nonoperable group of craniocerebral injuries and who has been adequately treated as such fails to improve or actually gets worse under that therapy. The history, symptoms, signs and condition of the cerebrospinal fluid are those of the associated nonoperable craniocerebral injury. The intracranial pressure may be high, normal or low. The pathology which includes that of the associated brain injury is that of a slowly expanding clot caused by bleeding from one or more ruptured veins in the cerebral subdural space (very rarely in the interhemispherical or cerebellar subdural space). The clot may be made up of either (a) pure blood that has the appearance of heavy machine oil, soft currant jelly or a mixture of the two and is commonly known as the "solid" type (this is the type that is caused by the rupture of a bridging vein, is not necessarily associated with any brain injury and is relatively rare) or (b) a mixture of blood and cerebrospinal fluid varying from mostly blood to mostly cerebrospinal fluid, with the blood either fluid or in the form of many small clots. This is commonly referred to as the "mixed" type. *Treatment* The subdural clot, regardless of type, should be removed as completely as possible through a subtemporal decompressive approach, the bleeding veins should be closed, the dura left open and the rest of the wound closed around one or more drains from the subdural space. Because these clots commonly occur bilaterally and because their presence is unrecognizable except by operative visualization of the cerebral subdural space, a bilateral exposure should usually be made. If the compressed brain does not expand when exposed at operation, Ringer's solution should be injected under pressure through a needle in the lumbar subarachnoid space, in quantities sufficient to expand the brain to normal size and shape (see page 12). If the cerebrospinal-fluid pressure remains below 150 mm. of cerebrospinal fluid after the wound is closed, and if the patient is properly hydrated, this injection may be repeated every twenty-four hours until the intracranial pressure is again normal. The amount of Ringer's solution to be injected is that sufficient to raise the intracranial pressure to 150 mm. of cerebrospinal fluid. Thereafter the treatment is as described under *Treatment Sections 1, 2, 3 and 4—Edema and Congestion* (pages 19 and 21) see also *Incisural Herniation* (page 38).

Subacute This differs from the preceding only in pathology and treatment. *Pathology* This is the same as that cited in the preceding section, except that the clots that consist of pure blood and the mixed hematomas that have sufficient blood in them have begun to organize by growing a neomembrane. This encloses the hematoma which at this stage usually resembles machine oil in consistence and appearance. *Treatment* If the patient's condition warrants it (which it usually does not) these clots should be approached through a bone-flap operation, because it is essential, if possible, to remove not only the clot but its neomembrane as well. Diagnostic bilateral transtemporal exploration is usually necessary first and can

be combined with a unilateral bone flap operation. Bilateral bone flap craniotomies should be separated by at least a week or ten days. Bone flap craniotomy is especially necessary in the newborn, because an intact neo-membrane will neither enlarge nor stretch with the growth of an infant's brain and, if not removed, will cause some degree of microcephaly by virtue of its compressive effect. If such a craniotomy cannot be performed because of the patient's precarious condition (this usually holds true in adults) the operation should be performed as described in the preceding section and attempts should be made to remove as much of the neo-membrane as can be reached. If the compressed brain does not expand when exposed in operation (see page 12) Ringer's solution should be injected under pressure through a needle in the lumbar subarachnoid space in quantities sufficient to expand the brain to normal size and shape. If the cerebrospinal fluid pressure remains below 150 mm. of water after the wound is closed and if the patient is properly hydrated this injection may be repeated every twenty-four hours until the intracranial pressure is normal. The amount of Ringer's solution to be injected is that sufficient to raise the intracranial pressure to 150 mm. of cerebrospinal fluid. Thereafter treatment will follow the methods described under *Treatment Sections 1, 2, 3 and 4—Edema and Congestion* (pages 19 and 21) see also *Intracranial Herniation* (page 38).

Chronic This differs from the preceding two types of hemorrhage in diagnosis, history, findings, pathology and treatment, despite the fact that every chronic subdural hematoma was at one time acute and passed unrecognized through the stages described above. The *diagnosis* is commonly the general one of brain tumor suspect, a chronic subdural hematoma being included among the possible choices. This working diagnosis is usually made, in part at least, as the result of ventriculography. The final identification of the clot as a clot can be made only by means of a craniotomy. The history is long, and the fact that the symptoms were originally associated with a blow on the head is either forgotten or considered by the patient to be of no moment. The *signs and symptoms* are those of a space-occupying intracranial lesion that has been expanding slowly over months or years. The *pathology* does not include that of the acute or subacute brain injury. The chronic clot, nevertheless, is the end result of the acute hematoma, just as cerebral scarring or adhesions are the end result of contusions or lacerations of the brain. The original clot has been composed of pure blood, is of the "solid" type and usually has been formed as a result of the rupture of a bridging vein rather than as the result of injury to the surface of the cerebrum. The solid chronic subdural hematomas may also eventually appear as an encapsulated collection of fluid with a high protein content, as a solid clot surrounded by a capsule and in the process of organizing, or as a fibrous thickening of the under surface of the dura. If it is not of these types it takes the form of an unencapsulated collection of subdural fluid that originated as a mixed hematoma in which the blood has been at a minimum and the cerebrospinal fluid at a maximum and which is now spoken of as a "fluid" type. It is these latter hematomas that are the most

by osmosis of cerebrospinal fluid through the arachnoid which acts as a dialyzing membrane. The unencapsulated chronic subdural hematoma and the encapsulated collection of fluid that is the end stage of an acute "solid" subdural hematoma are still not recognized as separate entities by many surgeons and are indiscriminately spoken of as "hygromas" or "hydromas." *Treatment* The chronic or end stage of the "solid" clot can only be properly treated by exposure through a bone flap operation. The clot and its capsule must be removed as completely as possible. The wound need not be drained. The postoperative therapy will conform to that required after removal of a cerebral tumor. This type of clot is almost always unilateral. The chronic or end stage of the dilute mixed hematomas, or "fluid" type, is best dealt with by transtemporal craniectomy which may need to be bilateral. The subdural space should be emptied as completely as possible and the wound or wounds closed about a drain from the subdural space. The drain should remain for thirty-six or forty-eight hours. If the compressed brain does not expand when exposed at operation (see page 12) Ringer's solution should be injected under pressure through a needle into the lumbar subarachnoid space in quantities sufficient to expand the brain to normal size and shape. If the cerebrospinal-fluid pressure remains below 150 mm. of cerebrospinal fluid after the wound is closed and if the patient is properly hydrated, this injection may be repeated every twenty-four hours until the intracranial pressure is normal. The amount of Ringer's solution to be injected is that sufficient to raise the intracranial pressure to 150 mm. of cerebrospinal fluid. The postoperative therapy should be that described under *Treatment Section 4—Edema and Congestion* (page 21) see also *Incurial Herniation* (page 38).

SUBPIAL HEMORRHAGE *Diagnosis* The diagnosis of subpial cerebral hemorrhage can be made only when the condition is exposed at operation. It occurs most commonly in association with either nonoperable craniocerebral injuries or other meningeal hemorrhages, although it may rarely be found alone at premortem examination. There is no particular history and there are no characteristic symptoms or signs of subpial hemorrhage. *Pathology* There is a hemorrhage which lies beneath the pia and which has dissected it free from the surface of the cortex for a variable distance. The contained vessels come with the pia. Other pathology is that of an associated craniocerebral injury. *Treatment* Too few cases have been seen and reported to have permitted anyone to devise a final method of treating this condition. Any individually indicated procedures must of necessity be purely empirical and should include therapy necessary in the treatment of any associated nonoperable craniocerebral injury as detailed above.

Compound Fractures of the Skull

In Table 3 are summarized the pathology condition of the cerebrospinal fluid, means of diagnosis symptoms signs and treatment of various types of compound fractures of the skull.

CASE	PATHOLOGY	CEREBRO-SPINAL FLUID	INTRACRANIAL PRESSURE	DIAGNOSIS MADE BY	SYMPTOMS	SIGNS	TREATMENT
1	Slowly expanding subcortical hematoma caused by a ruptured subcortical capillary degree of associated brain injury There may or may not be a fracture	Usually bloody	Increased	Exploratory Diagnostic trepanation	Those of the associated brain injury together with those of either extra- or subdural hemorrhage	Those of the associated brain injury together with those of either extra- or subdural hemorrhage	Operative removal of clot and subtemporal decompression
2	Lacerated wound of scalp fracture of skull any type of brain injury any type of cerebral hemorrhage bacterial contamination of all these layers fragments of bone and foreign bodies in brain substance	Clear or bloody	Increased	Palpation to the wound by the operator's sterile finger	Those of the associated brain injury potential or actual meningitis or ventricular or meningeal hemorrhage later those of encephalitis or brain abscess	Those of the associated brain injury those of ventricular or meningeal hemorrhage later those of encephalitis infection	Minimal handling at the first dressing dressing within 48 hours or sooner removal of all loose bone clots of scalp and dressings of scalp drainage treatment of associated meningeal hemorrhage brain injury chemotherapy and antibiotic therapy
3	All or none of the above comminution between the bone and intracranial cavity	As above	Normal or increased	X-ray examination	Those of the associated brain injury meningitis, encephalitis, brain abscess or osteomyelitis	Rhinorrhoea, proptosis, exophthalmos, those of the associated brain injury or meningitis	As above except that the wound is dressed with antiseptic and obliteration of the frontal sinuses if involved
4	All or none of the above comminution between the bone and intracranial cavity	As above	Normal	Demonstration of rhinorrhoea	Constant watery discharge from nose or into nasopharynx, those of meningitis	Rhinorrhoea, those of any associated brain injury or meningitis	Plastic closure of the nasal and ethmoidal ostia-pouching by way of a transfrontal craniotomy
5	All or none of the above comminution between the car and intracranial cavity	As above	Increased	Demonstration of C.S.F. escaping from the ear	Those of the associated brain injury with or without meningitis	Otorrhoea, often bloody those of any associated brain injury or meningitis	Maintenance of normal intracranial pressure by ventricular drainage no operation, maintenance of free drainage from the ear
6	Any type of brain or cranial injury local cerebral compression There may or may not be a scalp hematoma	Clear or bloody	Increased	X-ray examination	Those of the associated brain injury compressive seizures, headache, paralysis later	Those of the associated brain injury compressive seizures, abnormal E.E.G., headache, paralysis later	Operative removal or elevation after the intracranial pressure has been returned to normal but not before

COMPOUND FRACTURE OF THE VAULT OF THE SKULL. The *diagnosis* is made by palpation through the lacerated wound of the scalp with one finger of either hand enclosed in a sterile glove. Except in gunshot wounds, neither diagnosis by x-ray nor inspection with or without the aid of instruments is adequate. The history *symptoms signs* and condition of the cerebrospinal fluid and intracranial pressure are those listed under *Nonoperable Craniocerebral Injuries*. There is in addition a lacerated wound or in gunshot wounds, one or more puncture wounds of the scalp and deeper tissues. *Pathology* Fundamentally the pathology of a compound fracture of the skull is that of a contused or lacerated brain, the bruise or tear of which has been contaminated by a variety of bacteria and complicated by the presence of a communicating, similarly contaminated contusion or laceration of the dura fracture of the skull and lacerated wound of the scalp. If steps are not taken to remove or control the contamination, it will change into infection by the end of forty-eight hours after its initiation, and ventriculitis, encephalitis, brain abscess, meningitis, subdural abscess, osteomyelitis and cellulitis or abscess of the scalp may be added. If the compounding extends through the vault, the original contamination will be from bacteria that were present on the surface of the structure that struck (or was struck by) and penetrated at least the scalp and skull. If the compounding was not of the vault but extended into one of the air sinuses or through the cribriform plate, the contamination will be caused by the bacteria that commonly inhabit the airways of the victim and if it extends into the inner ear middle ear or mastoid cells the contamination will be through the bacteria that commonly inhabit the cavities of the ear and mastoid and, possibly the nasopharynx of the victim. If the compounding was the result of a bullet, gunshot, high-explosive-shell fragment, shrapnel or similar wound, the bacteria that contaminated the wound will be those that were present on the hair and on and in the scalp as well as those that were on and in any pieces of head covering or other foreign material that was either carried or blown into the wound. This gunshot type of compound fracture is further complicated by the presence of multiple fragments of devitalized bone that have been exploded down and into the tissues surrounding the missile tract. The missile itself may be considered to have been sterilized. Prognosis and treatment are greatly influenced by the depth of the compounding and any involvement of the ventricles, the first because of the associated distribution of the bacteria and the second because of the technical difficulty of removing by mechanical means the bacteria that have been deposited in the deeper layers of the wound. In addition to the pathology characteristic of the compound fracture, any such wound may be accompanied by the pathologic changes characteristic of any of the other nonoperable and operable craniocerebral injuries. *Treatment* Without a clear understanding and appreciation of the pathology adequate treatment of compound fractures of the vault is impossible. After the diagnosis is made the first step is proper treatment of the scalp wound. This is conditioned by the fact that during the first forty-eight hours the wound is contaminated and not infected, and

further by the fact that, if they are undisturbed sufficient tissue barriers may exist to keep this contamination within the bounds limited by the physical contact between the contaminating agent and the craniocerebral tissues. Extensive manipulation and irrigation or washing of the wound destroy these bounds and spread the contamination farther than it would otherwise have extended. Until and unless the surgeon is prepared to proceed in a matter of minutes to limit mechanically such a spread, his therapy of the wound must be gentle enough not to produce it. On the other hand, the use of chemotherapeutic and antibiotic agents is essential as an aid in keeping contamination from becoming infection. Consequently the wound must be manipulated sufficiently to insure the introduction of these therapeutic agents.

To accomplish these conflicting ends, no more should be done at the first dressing than to cut off (but not shave) enough hair to expose the superficial aspect of the wound or wounds in all its parts. Branches of the superficial temporal, occipital and supraorbital arteries that are bleeding may require tying, but all other bleeding can and should be controlled by pressure. The wound is then frosted with sulfanilamide powder and a dry sterile dressing applied under an elastic Ace bandage. At the same time surgical shock, if present, is treated with a transfusion of 500 cc. or more of whole blood, any less important injuries are cared for on a temporary basis, a lumbar puncture is done, the type of nonoperable complicating cerebral injury that is present is diagnosed and treatment for this latter started in accordance with the data given under the appropriate headings above. Sulfadiazine in sufficient amounts to maintain a level of 10 to 15 mg. per 100 cc. in the blood is started by mouth or intravenously and penicillin is given intramuscularly at the rate of 100 000 units every six hours and intrathecally with each lumbar puncture at the rate of 10 000 units every twelve hours.

As soon as the patient is no longer in surgical shock and is in sufficiently good general condition to withstand the major operation that may be necessary and providing he is in a large well equipped hospital with a competent, well trained surgical staff and providing adequate amounts of whole blood for transfusion are available as needed—all of which conditions are usually not fulfilled or fulfillable for twenty-four hours after the accident—and in any event within forty-eight hours or in cases of extreme urgency seventy-two hours after the accident, the patient should be taken to the operating room for a formal débridement and primary suture of his wound (see page 227). (The presence of a rapidly developing meningeal clot will supersede other conditions and will change accordingly the preoperative requirements as given.) Postoperative care should include the continued administration of sulfadiazine by mouth (unless renal complications or sensitivity develop) and intramuscular and intrathecal administration of penicillin until the infecting organism has been isolated from the culture and its sensitivity toward the antibiotics in general determined. Thereafter the particular antibiotic or chemotherapeutic agent that is most effective

against the specific infection should be used as indicated until all danger of central-nervous-system infection is past. Otherwise, treatment should follow the directions contained in *Treatment Sections 1 2 3 and 4—Edema and Congestion* (pages 19 and 21)

COMPOUND FRACTURES INTO THE FRONTAL SINUSES. The *diagnosis* is made from x-ray films. The history *symptoms signs* and condition of the cerebrospinal fluid and intracranial pressure are those of the nonoperable or other operable craniocerebral injury that may be present. In addition, there may be a history of a discharge of cerebrospinal fluid and excessive bleeding from the nose, and the appearance of marked swelling and ecchymosis about the forehead and orbits. The *pathology* is as described under *Compound Fracture of the Vault* except that there may be no external wound and a pneumoencephalocele may be present. The *treatment* varies with the amount of damage to the sinuses. If the fracture is limited to a linear crack of the posterior wall with minimal or no displacement of the bone fragments and there has been so little bleeding from the nose or into the periorbital tissues that one may conclude that the mucous membranes of the sinuses have probably not been torn the treatment is limited to that suitable for the associated nonoperable brain injury. It is described under *Treatment Sections 1 2 3 and 4—Edema and Congestion* (pages 19 and 21) together with the administration of sulfadiazine penicillin and other appropriate drugs as described under *Compound Fracture of the Vault*. An associated pneumoencephalocele requires no therapy of itself but does cast doubt on the integrity of the sinus mucous membrane. If the fracture of the posterior wall of the sinus is so comminuted and has such displacement of the fragments or is otherwise so extensive as to warrant the belief that the mucous membrane has been so torn as to permit free communication between the cavity of the nose and the meningeal spaces, or if the fracture of the frontal sinus is part of a compound fracture of the frontal bone nonoperative therapy will be inadequate and débridement must be performed.

The rules governing the optimum time of operation preparation of the field and the technic of débridement are those listed under *Compound Fracture of the Vault* except that this compound fracture must always be drained because complete mechanical sterilization of the wound is out of the question on account of the impossibility of closing off the nose. Care must be taken also not to remove the supraorbital ridge during the débridement of the bone because of the resultant cosmetic deformity. All "bone pockets" must be eliminated by flattening the frontal sinuses against their anterior wall. This can only be accomplished by completely excising the posterior walls and by removing all the mucous membrane. Postoperative therapy includes adequate oral administration of sulfadiazine and intramuscular and intrathecal administration of penicillin and other appropriate drugs until danger of infection is past, in addition to the treatment outlined under *Treatment Sections 1 2 3 and 4—Edema and Congestion* (pages 19 and 21).

COMPOUND FRACTURE OF THE CRIBRIFORM PLATE AND COMPOUND FRACTURE OF THE PARANASAL SINUSES OTHER THAN THE FRONTAL SINUSES

Diagnosis This is made on the basis of a history of a craniocerebral injury and the demonstration of a cerebrospinal rhinorrhea. The history *symptoms signs* condition of the cerebrospinal fluid and intracranial pressure are those of the associated nonoperable cerebral injury. In addition there will be a persistent constant flow of cerebrospinal fluid from the nose or into the pharynx. *Pathology* In addition to the pathology of whatever nonoperable craniocerebral injury may be present there is a communication between the nose and the cerebral subarachnoid space by way of a patent funnel of dura and arachnoid that is incorporated in a fracture of the cribriform plate or in the wall of one of the ethmoid or sphenoid sinuses. As a result, there is a constant escape of cerebrospinal fluid from the cerebral subarachnoid space. The latter may not be immediately evident however if the patient is lying on his back. In this position the cerebrospinal fluid runs into the nasopharynx and is swallowed. It will be necessary in many instances, therefore to sit the patient erect in order to demonstrate the rhinorrhea. *Treatment* Early closure of the fistula is essential. This can only be carried out by exposing it through an osteoplastic frontal craniotomy done on the right in right-handed patients and on the left in left-handed patients. When the fistula is thus exposed extradurally it is amputated close to the cranial surface of the bone the hole in the arachnoid is closed with a piece of "foam" and that in the dura with a piece of "foam" or pericranium. If necessary a free graft of dura may be stitched in place if the dural defect is large. The amputated portion of the fistula that lies against the bone is coagulated and it and the fracture line are sealed with "foam". The bone flap is replaced after leaving 50 000 units of penicillin in the wound the bone and scalp tissue are frosted with sulfanilamide powder and the wound is closed without drainage. The postoperative treatment is the same as that described under *Compound Fracture of the Vault* and under *Treatment Sections 1 2 3 and 4—Edema and Congestion* (pages 19 and 21).

COMPOUND FRACTURES INTO THE INNER AND MIDDLE EAR, THE MASTOID CELLS AND THE PETROUS PORTION OF THE TEMPORAL BONE. *Diagnosis* This is made from x-ray films, the presence of Battle's sign, a history of a craniocerebral injury and a cerebrospinal otorrhea. The history *symptoms signs* condition of the cerebrospinal fluid and intracranial pressure are all those appropriate to the associated nonoperable craniocerebral injury. In addition there is a constant flow of cerebrospinal fluid from the external ear. This is mixed at first with a considerable amount of blood which gradually clears up. Bleeding from the external ear without any admixture of cerebrospinal fluid is not evidence of compounding through the ear. *Pathology* In addition to the pathology characteristic of the associated nonoperable cerebral injury there is a linear fracture in one of the bones adjacent to the ear. This has caused a tear in the arachnoid and dura that coincides with the fracture line in such a way as to permit cerebrospinal fluid to leak directly or through a ruptured drum into the external ear and thus establish a com-

munication between it and the meningeal spaces. *Treatment* This is almost exclusively nonoperative. Its aim is to maintain the cerebrospinal fluid at a pressure low enough to permit the arachnoid to seal itself off. It is accomplished by lumbar drainage which is carried out twice daily. Sufficient cerebrospinal fluid is removed at each puncture to reduce and, if possible, to maintain the intracranial pressure at 100 mm. of water or less until the otorrhea ceases. The otorrhea of itself is not sufficient to maintain a sufficiently low cerebrospinal-fluid pressure to assure closure of the arachnoid tear without the help of the mechanical drainage by lumbar puncture. In addition, the patient is kept with his head elevated. In particular great care must be taken to see to it that nothing interferes in any way with the flow of blood or cerebrospinal fluid from the external ear. The ear should never be plugged but should be covered with a flat piece of sterile gauze held in place with adhesive plaster on the hair and jaw. For the rest, the treatment described in *Treatment Sections 1 2 3 and 4—Edema and Congestion* (pages 19 and 21) should be used as appropriate and necessary.

Herniation of the Temporal Cortex through the Incisura of the Tentorium Cerebelli with Compression of the Brain Stem and Adjacent Structures

In Table 2 (pages 28 and 29) are summarized the pathology condition of the cerebrospinal fluid and intracranial pressure means of diagnosis, symptoms, signs and treatment. The diagnosis is made by exploratory operation and the release of at least 25 cc. or more of cerebrospinal fluid that has been trapped under pressure beneath the tentorium. This requires a stab wound of one leaf of the tentorium and produces a consequent lowering of the supratentorial pressure after which it may be possible to demonstrate the herniation of the temporal cortex through the incisura. The continued escape of cerebrospinal fluid and the gradual reduction of the hernia then relieve the antecedent compression of the brain stem. The diagnosis is suspected whenever 1 A meningeal clot (either extradural or subdural) has been previously removed and the patient fails to show signs of improvement in forty-eight hours or is definitely worse twenty-four hours after the operation 2 a negative diagnostic exploration for a meningeal clot has been carried out but the tentorium has been neither exposed nor incised as a part of the exploration and the patient fails to improve or gets worse in the twenty-four hours immediately after the operation 3 a patient who has been diagnosed as a subdural hematoma suspect presents any of the signs of brain-stem compression listed below 4 a patient who regardless of the diagnosis or treatment, is known to have sustained a craniocerebral injury suddenly shows a significant change for the worse as well as two or more of the signs and symptoms of brain-stem compression noted below 5 a critical lumbar puncture has been performed (see page 19)

Except for the signs and symptoms peculiar to compression of the brain stem and its neighboring structures, the history signs and symptoms of herniation of the temporal cortex through the incisura are those of the associated nonoperable or operable lesions that precede or accompany them.

In addition it appears, however that to produce an incisural herniation there must be an increase in the solid contents of one temporal fossa of the skull. As a result the brain moves laterally the temporal cortex is forced first into and then through the incisura and then if the patient survives the medial surface of the cerebrum herniates through the hiatus in the falx. Thus venous congestion and more edema are produced together with compression and reduction in size of the ipsilateral ventricle and subarachnoid space (Fig. 4). This in turn limits the size of the cerebrospinal fluid reser-

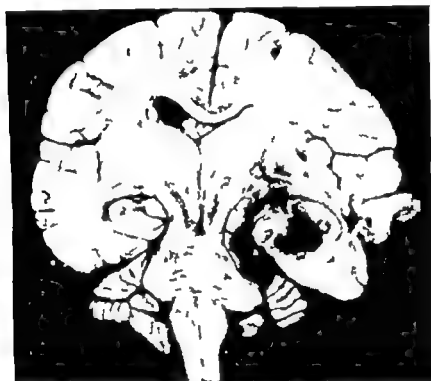


Fig. 4 Coronal section of brain showing incisural herniation of a part of one temporal lobe, as well as distortion and compression of the brain stem. (Courtesy of Dr Raymond D. Adams.)

voir as well as the area from which the fluid is absorbed. A secondarily resultant relative and actual increase in the cerebrospinal fluid contents of the cranial cavity is thus produced. The herniation is therefore likely to be associated with edema whether or not complicated by contusion or laceration (Fig. 1 page 8) and with meningeal clots above the tentorium (Fig. 3 page 27). In particular those clots that are located in the temporal fossa are most apt to be found accompanying incisural herniation. The signs and symptoms of compression of the brain stem and its neighborhood structures vary widely with the size and the acuteness of onset of the herniation as well as the rapidity and completeness of the blockade of the flow of the cerebrospinal fluid and its resultant trapping beneath the tentorium. Other factors are the effectiveness of the upward counter-pressure exerted

by the fourth ventricular and spinal-cerebrospinal-fluid column and the resistance to downward pressure by the structures in the posterior fossa and the foramen magnum. Early symptoms are an increasing stupor gradually deepening into coma, a hemiparesis, especially if it seems to be ipsilateral with a clot that has already been located, and increasing confusion. Later there may be convulsive seizures, hemiplegia and decerebrate fits. The signs include a third-nerve palsy and in particular unilateral or less frequently bilateral dilation and fixation of the pupils a stiff neck Magnus-de Kleijn reflexes of varying degrees decerebration, respiratory irregularity periodicity and increase in rate pulse irregularity and increase in rate, and blind



Fig. 5 Thrombosis, hemorrhage and edema of the brain stem, caused by incisional herniation of a part of one temporal lobe (Courtesy of Dr Raymond D. Adams.)

ness in half the visual field. In the latest stages of the herniation there may be a demonstrable lateral distortion of the third ventricular and hypothalamic structures by an associated movement of the supracallosal surface of one cerebral hemisphere through the opening beneath the falx cerebri. The precipitation of these signs and symptoms in close association with lumbar drainage of the cerebrospinal fluid from below the tentorium and the reduction thereby of a high subtentorial intracranial pressure to a normal or subnormal level as well as the sudden demonstration of a normal or low intracranial pressure by lumbar measurement in the face of adequate hydration, is a frequent but by no means invariable finding. This is especially true when there are clinical signs and symptoms that suggest an increase rather than the demonstrated lowering of the cerebrospinal-fluid pressure and when previous measurements of the cerebrospinal pressure have heretofore been consistently high. *Pathology* After a shift toward the less damaged side, the uncus and a variable amount of adjoining temporal cortex—possibly from both temporal lobes but usually from only one—

herniates through the incisura. The herniated portion becomes edematous and thrombosed and makes increasing pressure on the brain stem and adjoining structures (Fig. 4). The subarachnoid space there is closed off. As a result there is at least a partial interruption of the flow of cerebrospinal fluid from the lateral ventricles to the areas from which it is normally absorbed. This traps cerebrospinal fluid below the tentorium, distends the contralateral ventricle or possibly both lateral ventricles, distorts the third ventricle and raises the intraventricular pressure. Because the borders of the incisura are fixed and inelastic, the brain stem is moved laterally and the oculomotor nerves—and especially the third cranial nerve on one side

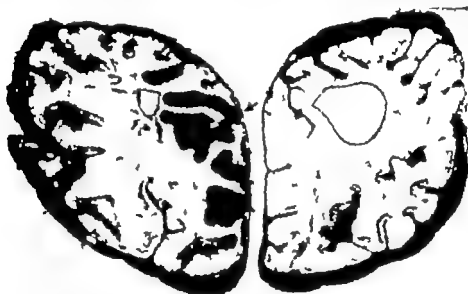


Fig. 6 Infarct of one occipital lobe, caused by compression of the posterior cerebral artery at the point where it had been caught in a herniation of part of one temporal lobe through the incisura. (Courtesy of Dr. Raymond D. Adams.)

—are then compressed. The aqueduct of Sylvius, being compressed, is narrowed, there is further distortion of the brain stem, edema develops and thrombosis and hemorrhages occur in the pons and midbrain (Fig. 5). The third ventricle and hypothalamic region are also distorted. Further overfilling of the incisura compresses the posterior cerebral artery and vein causing edema, thrombosis, hemorrhage and ischemic necrosis of the corresponding occipital lobe (Fig. 6). It is probable that the head of pressure developed in the lateral ventricles is further increased by their compression and the reduction of their capacity because of the associated cerebral edema. This keeps the aqueduct sufficiently open to permit some flow of cerebrospinal fluid until very late in the process and thus permits an operative correction of this otherwise lethal mechanism but only after a degenerative encephalopathic but nonfatal decerebration has taken place. *Treatment* The only effective treatment is surgical but this is effective only when done at the earliest possible moment. It must include release of the trap

subarachnoid space either on the surface or in one of the sulci, by a small outlet that may be sealed at the time the clot is looked for. *Treatment* This is operative when and if the diagnosis is made. The approach is usually through a transtemporal exposure; the clot is usually located by needling the cortex, after which it is usually removed by suction through a transcortical incision. If the bleeding vessel can be found (and it usually cannot) it should be closed. Unless other acute pathology is present the wound, including the dura, is closed without drainage. Later treatment, if the cause has been a craniocerebral injury, is carried out in accordance with the directions listed in *Treatment Sections 1, 2, 3 and 4—Edema and Congestion* (pages 19 and 21). If the pathology is that of arteriosclerosis, berry aneurysm or other congenital vascular anomaly appropriate postoperative and convalescent care, including any necessary further surgery, should be provided after localization of the probable source of the hemorrhage by arteriography if that is indicated.

Post Traumatic Cerebral Atrophy

Diagnosis This is made by a history of a craniocerebral injury one year or more previously and the demonstration by pneumoencephalogram of significant cortical or subcortical cerebral atrophy. The history will be that of a craniocerebral injury accompanied by unconsciousness and amnesia and usually inadequately treated from the points of view of diagnosis and intracranial pressure. The *symptoms* will be those of the so-called post traumatic state or neurosis with emphasis on headaches. There will be no characteristic *signs* other than the cortical or subcortical atrophy with enlarged but normally placed and undeformed ventricles. The cerebrospinal fluid will be normal and the intracranial pressure often below 100 mm. of water. *Pathology* This will consist of a moderate atrophy of all the layers of the cerebrum resulting from an earlier mild, persistent and uncorrected increase in intracranial pressure with compensatory dilatation of the ventricles and cerebral subarachnoid space. In infants, unintentional placement of injected air in the subdural space may give the appearance of cortical atrophy when it is actually not present. Such forcible separation of the cortex from the dura may rupture a bridging vein and produce an unexpected acute subdural hematoma. This will produce appropriate symptoms and will require therapy that may even have to include a craniotomy. *Treatment* A subtemporal decompression over the nondominant hemisphere with emptying, so far as possible, of any exposed "puddled" areas of cerebrospinal fluid in the subarachnoid space, should be carried out. The wound should be closed except for the dura, which must be left open around a drain from the subtemporal subarachnoid space. The drain is left in place for thirty-six to forty-eight hours. Postoperative and convalescent treatment is carried out in accordance with the methods described in the appropriate paragraphs of *Treatment Section 4—Edema and Congestion* (page 21). This condition is uncommon, and predictions as to the efficacy of this therapy must be guarded.

Depressed Fracture of the Skull

In Table 3 (page 33) are summarized the pathology condition of the cerebrospinal fluid means of diagnosis, symptoms signs and treatment of depressed fractures of the skull. The *diagnosis* is made by stereoscopic x-ray examination of the skull and a history of a craniocerebral injury. The history *symptoms signs* and the condition of the cerebrospinal fluid and intracranial pressure conform to the similar data described under either *Non operable Craniocerebral Injuries* or *Meningeal Hemorrhages* or both. In addition there will be x ray evidence of a depression of the inner table or of both tables of the skull. The depressed area is usually covered by a subperiosteal or subgaleal hematoma. It is impossible, when it occurs alone to certainly differentiate such a hematoma from a depressed fracture except by x-ray *Pathology*. In addition to the pathology characteristic of whatever type of nonoperable craniocerebral injury or meningeal hemorrhage that may be present, a variable number of fragments of bone will be found extending in a wide variety of ways into the cranial cavity where they do not belong. As a result, there may or may not be tears of the dura, of any of the venous sinuses or their large tributary veins, of the middle meningeal vessels or of the cortex and its vessels. The torn vessels may not bleed because they may have been plugged by a bone fragment. Fragments of bone that are depressed and compounded are considered to be compound fractures from the points of view of diagnosis and treatment. *Treatment* The most important part of the treatment of a depressed fracture of the skull is making certain that any increased cerebrospinal fluid pressure, with its associated increased intracranial venous pressure and congestion, has been permanently restored to normal before any operative attempt is made to elevate or remove the depressed fragment or fragments. Only in this way will the danger of an unnecessary death from operative hemorrhage or air embolus, an almost inevitable accompaniment of the removal of any depressed fragment that has hitherto been plugging a torn congested sinus be avoided. This reduction of the intracranial pressure to normal is accomplished by the treatment outlined in *Treatment Sections 1 2 and 3—Edema and Congestion* (page 19). At operation the depressed fragments of bone are removed, the dura and any ruptured vessels are closed if they have been torn, all clots are removed and hemostasis is obtained, after which the scalp is closed in layers without drainage. The depressed fragments are always much larger than they seem by x-ray. Depressed fractures that have been allowed to remain unoperated upon long enough to allow the fragments to become united in their abnormal position are best dealt with by turning an osteoplastic flap with the depressed area in its center. The depressed part is then removed and the inner surface of the bone flap is smoothed over with an electric drill. In these long-standing cases the dura should be opened, any corticodural adhesions separated and the cortex covered with "foam." The bone-flap is then replaced and fixed with two or three tantalum wire sutures to the adjoining bone and the scalp is sutured in layers without drainage.

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Cranioplastic Repair with Removal of Adherent Cortico-Dural and Cortico-Scalp Scars

Diagnosis This is made by x ray studies and a pneumoencephalogram. There will be a history of an old cranioerebral injury or craniectomy with local loss of bone as the result of the injury or through operative removal. The *symptoms and signs* will vary from a psychoneurosis based on fear of another injury or of distress at the disfiguring cosmetic effect, to those characteristic of persistent cortical irritation, and they will range from headaches and dizziness to convulsive seizures. The cerebrospinal fluid and intracranial pressure will be normal. **Pathology** This will vary from adhesions between the edge of the bone defect and the underlying dura to extensive scarring involving the scalp, bone, dura and cortex with degenerative intracortical cysts and other changes. There may be distortion of the underlying ventricle toward the scar and enlargement of the opposite ventricle. **Treatment** The bone defect can be closed by shaped tantalum or acrylic-resin plates² held in place by screws placed in normal adjoining bone through tabs on the edge of the prosthesis. The plates may be shaped before operation on dental-compound molds or the simpler ones may be cut and shaped during the operation. In the latter instance the tantalum is molded by hammering it on a steel block with a ball-peen hammer and the acrylic resin is shaped by molding it in hot sterile water. Before the plates are applied the bone edge should be cut back slightly, the dura freed from it and the underlying cortex and any dural defect covered with "foam," or possibly with polythene film or a nylon sheet such as is made by DuPont. Closure of the dural defect by fascia lata transplant, Fibrin film, Cargile membrane or Amniotin should not be attempted, because these structures serve merely as a base for a new and equally disabling scar that will adhere again to the underlying cortex. Free periosteal grafts, applied with their outer surface innermost, may also be tried.

It is important that the cortex be completely freed of its attachments to the dura at all points and that any fibrous scar in it be removed as completely as possible. Identification of the motor cortex by electrical stimulation prior to any cortical ablation is essential, and great care should be exercised that only minimal damage is done to the adjoining cortex. The scalp defect should be closed without tension and with a linear suture line over any implant. If necessary a flap from the adjacent scalp can be rotated in such a manner as to place the suture line at one side of the plate. The wound must be closed without drainage. Postoperative collections of fluid other than cerebrospinal fluid between the scalp and the plate should be removed by tapping and suction through the scalp. Cerebrospinal-fluid collections can only be obviated by reoperation and closure of any arachnoidal fistula after excision of any associated cyst lining. If the wound becomes infected the implant should be removed at once. None of these cranioplasties should be performed until there is no further danger of infection from any bacteria that were originally present in the wound and have remained dormant after the earlier injury or operation. Acrylic resin—and especially metallic—plates

should never be used to cover a defect in which there is still an open communication with the nose either directly or by way of an air sinus. Such fistulas must be permanently sealed. If necessary the frontal sinuses in particular should be obliterated before a cranioplastic closure is done. If these precautions are not taken the incidence of wound infection with resultant later necessary removal of the plate occurs in an unjustifiably high percentage of cases. Bone chips, placed extradurally, have been used to close defects in this area.² Postoperative and convalescent treatment will follow the lines laid down in *Treatment Section 4—Edema and Congestion* page 21.

Cranioplastic Repair for Symptoms Produced by Deficiencies of Bone on Opposite Sides of the Skull

Diagnosis. Under certain circumstances it may become necessary to produce by surgical means bony deficiencies opposite to each other in the skull. The presence of such openings will of itself produce symptoms that can be corrected only by the closure of one or both of the openings and that occur regardless of the absence of any other pathology in the central nervous system. The symptoms are all subjective and are usually described as dizziness on change of position, dizziness on getting out of bed in the morning, a feeling of instability inability to take very active exercise or to do any work, no matter how simple and a "feeling as though the brain were loose" or "were shaking." The symptom complex is difficult to distinguish from that of post traumatic state or neurosis (see page 48) indeed, the former is usually accompanied by some degree, at least, of the latter. Without cranioplastic repair the symptoms plus the neurosis will become permanent and completely disabling in so far as productive activity is concerned. The longer the postponement of necessary repair the less complete is the patient's recovery from both the symptoms and the neurosis. **Pathology.** The creation of opposite cranial defects, especially if the dura has been left open, subjects the brain to the direct effect of atmospheric pressure removes its lateral supports, causes it to shift with any change of body position and produces a mechanically unphysiologic condition. There need be no actual damage to the brain itself to produce disabling symptoms in such cases. **Treatment.** The treatment of this condition is to close one or both of the cranial openings and, if possible any dural defect as well. The technical details of this procedure are fully covered in the previous section, *Cranioplastic Repair with Removal of Adherent Cortico-Dural and Cortico-Scalp Scars* (page 46) which should be consulted.

Post Traumatic Cortical Scars with Convulsive Seizures

Treatment of such scars by surgical excision as an adjunct to anticonvulsant medication has not yet become sufficiently standardized to be undertaken, except in one or two clinics by a limited number of specially trained neurosurgeons. The necessary equipment—notably the electroencephalographic portion—is extensive expensive and difficult to operate. Too few

patients have been operated upon and none have been followed for sufficient time to permit judgment of the long-term value of this surgical procedure

The Post-Traumatic State or Post Traumatic Neurosis

Certain patients who have sustained significant cranio-cerebral injuries years before they are seen by the surgeon will complain of disabling symptoms that allegedly prevent them from doing gainful labor or its equivalent. Before treatment can be prescribed for them the surgeon must be certain that the symptoms have an organic—as opposed to a psychosomatic—basis and that the symptoms are actually the result of the earlier cranio-cerebral injury as claimed, and not traceable to some as yet unrecognized or concealed intercurrent disease. All such patients should therefore be hospitalized and carefully studied in order to settle these points. Any demonstrable organic disease whether of the brain or of any other organ, should be appropriately treated forthwith. If it can be shown that there is no organic cause for the patient's complaints, an attempt should be made to straighten out his psychosomatic disorder. Fundamentally these patients complain of symptoms in order to justify their continuing fear of death, invalidism or insanity and because their initiative has been sapped to the vanishing point by the efforts of friends and relatives to keep them from exerting themselves. This belief in the necessity for nonexertion arises from the mistaken idea on the part of their physician that the convalescent care of cranio-cerebral injuries requires virtually complete physical rest for the patient, and from the emphasis frequently laid by their lawyers on the importance of maintaining disabling symptoms until all litigation has been ended. As a result, the patients, especially if they have a pretraumatic instability or psychoneurosis, exaggerate to themselves as important or even lethal such ordinary symptoms as an occasional headache, attacks of dizziness, or fatigue and the like. They refuse to make any decisions about their own welfare preferring to vegetate. In consequence their efficiency and general condition especially as regards their physical reserve, circulation and muscular activity reach a low level. Treatment must be directed first toward removing their fears. This is best accomplished by the hospitalization and study referred to above, which must be thorough. The next step is to redevelop their initiative and at the same time to return their physical condition to normal. These two procedures go hand in hand and are accomplished by setting up for them a general program of regular gradually increasing physical exercise, the carrying out of which, as regards both decision and details, must be left strictly to the patient. As a corollary to this, it will be at once apparent to the patient that whether he gets better stays the same or gets worse it will be strictly through his own efforts and decision. Moreover any consequences of these efforts such as regained ability to do work and to lead the normal social life that has hitherto been (as he and his friends and family believed) denied him will be either restored to him or further denied him solely as the result of his own personal unassisted efforts.

The details of this therapy are outlined in *Section 4—Edema and Congestion—Convalescent Care* (page 21). If this program is to be successful there must be co-operation by the insurance company, the employer, the patient's lawyer and the patient's family. The patient himself must have sufficient intelligence to grasp the necessary intellectual implications and should understand his own problem thoroughly. The longer the period between the injury and the start of this regimen, the poorer are the chances of rehabilitating these patients.

The Treatment of Cranocerebral Injuries in the Newborn

All such patients must be hospitalized for treatment.

General Considerations

Although in general the basic factors that govern treatment of cranio-cerebral injuries in the newborn are the same as those that apply in the adult injuries, there are certain fields in which the details differ. These differences must be appreciated and adjustments must be made for them if the best results are to be obtained.

ETIOLOGY In addition to the damage caused by the application of violence directly to the skull and its contents, the effect of anoxia must be reckoned with. The former type of damage is represented by depressed fractures of the skull, meningeal tears, meningeal hemorrhages and cephalhematomas. They differ in no important pathologic essential from similar injuries in the adult. The cerebral changes produced by the anoxia of asphyxia appear to be peculiar to the newborn, however. Aside from such obvious causes as near-strangulation from the wrapping of the umbilical cord around the neck of the fetus and separation and infarction of the placenta, the asphyxia and hence the anoxia and neural cell damage arise out of the processes of labor itself. This source may be hidden because lethal anoxia may be entirely (and is frequently partially) in effect while the fetus is still in the uterus and during the initial stages of labor. The effects are weighted not on the side of immediate mortality but rather on the side of late extremely disabling morbidity. Such conditions as porencephaly, hydrocephalus *ex vacuo*, epilepsy, idiocy, any degree of spastic paralysis or paresis or Little's disease may be the direct results of such oxygen deprivation. Predisposing factors are abnormally precipitate or long labors, maternal anesthesia, the use of certain respiratory-depressant drugs, dry labor, cephalopelvic disproportion with arrest of the descent of the fetal head, prolongation of the second stage of labor or excessive molding of the fetal head. In many of these the factor of trauma added to an already existing anoxia enhances the effect of the latter. Thus, in estimating the cause of cerebral injury in the newborn these various possibilities must be given due consideration.

Another factor peculiar to the newborn is the small amount of energy reserve that is present in this group of patients. This is tapped to the utmost by the attempts of the organism merely to maintain life. Such added expenditures as the baby's response to handling, to dehydration and to the

energy necessary to suck nourishment from either the mother's breast or an artificial nipple on a bottle may under such critical circumstances, be more than is compatible with life. This is true even in otherwise robust, normal babies born at full term. It is even more true in those born prematurely. Any treatment of these invalids must be given with these various factors taken into account, and surgery and anesthesia in particular must only be undertaken with a full appreciation of the extra risk involved.

PROPHYLAXIS Finally prophylaxis against cranlocerebral injuries in the newborn is not a matter of being ready to deal with those emergencies that manifest themselves during labor and immediately postpartum but, rather is found in the understanding of the cause of such emergencies and, therefore of the proper management of labor from the first months until after the umbilical cord is divided.

The Asphyxial Type of Intracranial Injury

The diagnosis is made by the birth history as indicated above and by the findings of an abnormally high intracranial pressure with bloody cerebrospinal fluid at lumbar puncture. It should be noted that the normal intracranial pressure in the newborn is lower than that of the child or adult. In the newborn it ranges between 50 and 100 mm. of cerebrospinal fluid, with the range usually nearer 50 than 100 mm. The signs and symptoms commonly are hypertonicity of the musculature in general, an abnormal or poor cry, cyanosis and failure to nurse either at the breast or from a bottle. The hypertonicity may be evidenced by a characteristic excessive muscular reaction to sudden sensory stimuli such as jarring of the crib or a loud noise. The cry may be absent, usually when associated with unconsciousness, may take the form of intermittent or constant shrieks, or may vary in volume, tone or frequency. Cyanosis will usually be associated with an abnormal respiratory rate or volume. Extremely sick babies may be pale, flaccid and apathetic, a condition that probably represents the imposition of surgical shock on the underlying intracranial pathology. These babies have an abnormal intracranial pressure and respond to the usual measures of combating shock. Except in this latter condition the fontanelle will usually be tight but is not necessarily so. The most reliable sign is an increase in intracranial pressure as measured on a manometer at lumbar puncture. This has been referred to above. Low intracranial pressures may have to be measured by a mercury manometer because less cerebrospinal fluid is needed to fill the system. The presence of respiratory oscillations is sufficient to assure the operator that his needle is in direct contact with the cerebrospinal fluid. **Pathology** The pathology is essentially that described under *Edema and Congestion of the Brain* (see page 19) plus hemorrhage into the perivascular and perineuronal spaces, beneath the pia and arachnoid and from the choroid plexus. If this is not treated, there may be ischemic necrosis of large areas of the cortical and subcortical structures, with the disappearance of considerable areas of cerebral tissue. According to the size of the destroyed areas there will be a varying functional loss, permanent in nature,

that may be manifested later on by anything from mental retardation, idiocy and spastic paralysis to porencephalic cysts and hydrocephalus *ex vacuo*. *Treatment.* If there is evidence that surgical shock is present, this must be dealt with first of all, in the customary way. Whole-blood transfusions, maintenance of body temperature, adequate fluid administration and uninterrupted complete physical immobility are essential. With the correction of surgical shock the next step is the reduction of any increased intracranial pressure that may be present. This is best accomplished by lumbar drainage of the excess cerebrospinal fluid, by means of daily lumbar punctures. Attention should be called again to the fact that the average normal intracranial pressure in the newborn is 50 mm. of cerebrospinal fluid and that it never under any normal circumstances exceeds 100 mm. of cerebrospinal fluid. Equal in importance to this direct treatment are the indirect procedures. These have to do with the baby's lack of energy and reserve. The baby should not be moved out of its crib and the necessary handling in the crib must be reduced to a minimum. Nursing from the breast is prohibited and nursing from a bottle is to be permitted only if the baby sucks energetically. If it does not, then it should be fed by a Breck feeder or from a medicine dropper. Fluids must be provided at the rate of 2 to 3 ounces per pound of body weight per twenty-four hours and should be given by mouth if possible, and if that is not possible by clysis. Oxygen may be given for cyanosis. Morphine is contraindicated. If the baby fails to respond to this therapy a ventricular puncture may be made through the anterior fontanelle to rule out ventricular hemorrhage. If this is present the ventricle may have to be repeatedly punctured in addition to whatever lumbar punctures are necessary. If the baby continues to do badly the presence of a subdural or extradural hematoma should be suspected and prompt measures taken to verify the diagnosis and, if necessary, to institute appropriate treatment.

The Traumatic Type of Intracranial Injury

This type of injury is always added to and complicated by the *Asphyxial Type* (see page 50). It is analogous to the operable group of craniocerebral injuries in the adult. Treatment rendered these lesions is given in addition to and does not replace, the treatment necessary for the asphyxial lesions. In this group are included tears of the meninges, ruptures of the venous sinuses, internal cerebral veins, bridging veins and meningeal arteries, simple and depressed fractures of the skull and cephalhematomas.

MENINGEAL TEARS, RUPTURES OF VENOUS SINUSES, RUPTURE OF THE VEIN OF GALEN AND RUPTURE OF THE INTERNAL CEREBRAL VEINS. These conditions are all caused by excessive molding of the head during labor or delivery. They are all associated with the *Asphyxial Type of Intracranial Injury* (see page 50). The diagnosis can only be made with certainty at autopsy. It can be inferred from the history, the shape of the head at birth, the amount of blood in the cerebrospinal fluid as collected through repeated lumbar punctures and the presence of surgical shock. The signs and symptoms are those of surgical shock added to any deformity of the

head that may be present, plus those of the *Asphyxial Type of Intracranial Injury* (see page 50) *Pathology* This varies to include asymptomatic tears in the substance of the falx and the leaves of the tentorium, splitting of the superior layers of the tentorium at its junction with the falx, and laceration of the superior sagittal, straight or lateral sinus or sinuses, and division of the vein of Galen or of one or both internal cerebral veins. All of these lesions are accompanied by the cerebral changes associated with asphyxia. Those pathologic states in which large venous channels have been ruptured are characterized, in addition by large intracranial collections of blood, particularly around the brain stem or in the ventricles, or both. *Treatment.* This is limited to that suitable for surgical shock and the cerebral changes produced by asphyxia and any possible late removal of residual subdural or extradural hemorrhages. Early surgical interference is impossible. Such severely injured patients as these commonly die shortly after birth, regardless of any therapy.

RUPTURE OF BRIDGING VEINS Under certain circumstances of molding in which there is marked displacement of the sagittal suture or of the squama and the parietal bones, there will be tearing of the bridging veins during their free course between the dural vessel and the cortical vessel. Depending on the point at which this tearing takes place, the resulting hemorrhage will be either subdural or subarachnoid. In either instance the meningeal hemorrhage will be accompanied and complicated by the associated *Asphyxial Type of Intracranial Injury* (see page 50) which of itself requires treatment in addition to that afforded the other condition. It is usually impossible, except at autopsy (and often not possible even then) to do more than infer the source of these hemorrhages.

CEREBRAL SUBDURAL HEMATOMA. The subdural hematomas in infants and the newborn are similar in almost all instances to the solid subdural hematoma, either acute or chronic in adults (see pages 30 and 31). The only significant differences are to be found in the method of making the diagnosis and the fact that in the newborn and in infants it is no longer optional but imperative to remove all of the neomembrane surrounding the clot. If this is not carefully done, the normal growth of the hemisphere covered by the membrane will be limited to the degree of stretch of the latter and will be materially retarded, with resulting marked functional loss later in life. The diagnosis is suspected when a baby who has been efficiently treated for the *Asphyxial Type of Intracranial Injury* and who is not toxic from dehydration no longer improves or starts to get worse. Verification of the presence of a subdural hematoma, instead of being made by a trephination, can be made by a transfontanelle puncture of the subdural space. Because these clots are particularly apt to be bilateral in the newborn, bilateral needle explorations must be done. If evidence of a clot is found, the intramembranous space may be emptied in part by suction on the needle thus relieving any acute situation. This treatment is not definitive or final, however and it must be followed by a bone flap craniotomy with

complete removal of not only the clot but all its limiting membranes. Bilateral craniotomies should not be performed less than ten days apart.

SUBARACHNOID HEMORRHAGE. This is entirely analogous to *Traumatic Subarachnoid Hemorrhage* in the adult (see page 24). It may be effectively treated by those methods that are used to treat the *Asphyxial Type of Intracranial Injury* (see page 50).

RUPTURE OF THE MENINGEAL ARTERIES. When an intracranial artery is ruptured in the newborn which is extremely seldom, it is the middle meningeal artery. This produces a temporal extradural hematoma that is no different in its effect from one in an adult and that requires the same diagnostic methods and has the same therapeutic indications. This has been adequately covered in the previous section under *Extradural Hemorrhage* (see page 26). Associated with the meningeal hemorrhage is always some degree of *Asphyxial Type of Intracranial Injury* that requires active treatment on its own account (see page 50).

Bone Injuries

Fractures of the skull in the newborn occur as the result of compression of the head by misapplied forceps, local damage by pressure against either the promontory of the mother's sacrum or her symphysis pubis in the course of labor or the use of the Scanzoni maneuver.

EGG SHELL FRACTURE. *Diagnosis.* This is produced by the tip of one forceps blade which has been incorrectly applied to the head of the fetus. *Signs and Symptoms.* A palpable crackling and possibly some local deformity will be noted at the point of fracture. *Pathology.* The break is similar to the seen in an egg shell when an egg is dropped on something solid. There may or may not be a complicating *Intracranial Injury of the Asphyxial Type* (see page 50). *Treatment.* The fracture of itself requires no treatment. An associated condition should be treated in accordance with the indicated diagnosis and pathology.

DEPRESSED FRACTURE. *Diagnosis.* This is produced most frequently by local pressure on the skull of the fetus during labor by the mother's sacral promontory or symphysis pubis. It may also be caused by misapplied forceps. *Signs and Symptoms.* There is a local celluloid-ball type of palpable and visible fracture of the skull at the point of injury. A cephalhematoma may also be present, and there may be some degree of the *Asphyxial Type of Intracranial Injury* (see page 50). *Pathology.* The fracture is of the celluloid-ball variety. *Treatment.* The treatment is operative. The scalp incision should be short and linear and the operation should be done under local anesthesia only but with full formality. A small hole should be drilled in the uninjured bone at the edge of the depressed fracture. This drill-hole is most easily made by rotating the point of a knife blade on the skull. The hole is then enlarged sufficiently to permit the insertion of a straight, narrow periosteal elevator until its end is opposite the deepest point of the depression. The dura is carefully and gently separated from the overlying

bone during this insertion. Care should therefore be taken to see that the elevator does not have to cross a suture line. With the inner end of the instrument in the proper position, pressure is made outward on the apex of the depression and the whole fracture is snapped into place. After ascertaining, by waiting a few minutes, that there is no significant extradural hemorrhage the scalp is closed in two layers without drainage. The traction outwards of these fractures by hooks driven blindly through scalp and bone is dangerous and unsurgical and cannot be countenanced.

FRACTURE OF THE BASE OF THE SKULL. This results from the use of the Scanzoni method of applying forceps. In this maneuver the forceps, after being applied with their front looking toward the fetal forehead, are used to rotate the head in such a way as to bring the forehead to the rear. This may involve twisting the fetal neck through an arc that may exceed 90 degrees. The forceps are then removed, reapplied in the proper position, and used to make traction on the head with it and the fetal neck in the twisted position. As a result it is rather commonly discovered that a fracture of the base of the skull, including the foramen magnum, has been caused. In addition to the tremendous danger inherent in thus deforming the head and neck relations, there is added, because of interference with the respiratory tree and compression of the venous channels leading from within the cranium, a high degree of *Asphyxial Type of Intracranial Injury* (see page 50). *Treatment* The best treatment is not to use the maneuver.

Cephalhematoma and Contusion of the Face and Scalp

Contusions of the face and scalp are caused by either the natural course of labor or by the misapplication of forceps. They are of no significance unless the facial nerve has been injured. The *treatment* of such an injury is highly individualized and must be chosen strictly in accord with the indications presented by each separate case. *Cephalhematomas* These are more properly *subperiosteal hemorrhages*. The bleeding is limited by the anatomical extent of the involved bone and does not communicate with the cranial cavity. It may reach a point at which the loss of blood into the subperiosteal space is sufficiently great to necessitate a transfusion, however. These hemorrhages must not be confused with the subgaleal edema of a *caput succedaneum*. Both conditions will absorb of themselves if left alone. In particular the cephalhematoma must not be needled. Enough bone may form on the surface of such a clot to give a sensation of "crackling" on palpation. The new bone formation will not proceed beyond this point, however.

The Late Effects

GENERAL CONSIDERATIONS The late effects of craniocerebral injury in the newborn will vary from a slight mental retardation through idiocy and spastic paralysis to the disappearance of large parts of the cerebral substance and substitution therefor of fluid in the form of porencephalic cysts.

or hydrocephalus ex vacuo. It is not certain that *all* the late changes, with their varied degree of functional loss, can be prevented by proper therapy given early enough because surgical possibilities are limited and the most that can be expected from them is the provision of a beneficial atmosphere in which those cells that might recover will be given the opportunity to do so and in which the undamaged cells will not be damaged. However excluding the congenital abnormalities properly timed and applied surgery in conjunction with properly conceived and executed obstetrics, can be expected to reduce—and in a limited series definitely has reduced—the amount of this tragic unnecessary invalidism to a reasonable figure. One of the duties of every obstetrician is to be familiar with this fact, and one of the duties of every pediatrician is to be familiar with the methods by which intracranial damage in the newborn can be recognized at once after birth and, if he is unable or unwilling to apply such methods and follow them with appropriate therapy then it is his definite responsibility to see that some physician or surgeon is called in who is ready able and willing. The general practitioner who is both obstetrician and pediatrician has double the responsibility of either and by virtue of that privilege he can do more than either in the field of prevention. No progress will be made in the prevention of these tragedies until every doctor who is in any way associated in his practice with such a handicapped child considers himself personally responsible for the invalidism and thereupon takes such educational and other steps as are necessary to prevent, in so far as he is able its ever occurring again.

Once the die is cast, however and the deficient and deformed child is brought to the surgeon for treatment, two things must be made crystal-clear to all concerned. The first is that any decision concerning therapy or prognosis in the individual case cannot be adequately made or be expected even to approach the facts until after the size and configuration of the brain have been visualized by pneumoencephalography. All other necessary means of investigation should also be used, but encephalography is fundamental and cannot be dispensed with. There may be some attached risk, but this must be accepted for the sake of the greater benefits to be gained.

MENTALLY DEFICIENT CHILDREN WITH A NORMAL SIZED BRAIN After it has been demonstrated by a pneumoencephalogram that the brain of such a child is of normal size and after psychiatric studies have shown that he is nevertheless mentally defective, such a child should be promptly institutionalized in a school devoted to the care of such defectives. The presence of other kinds of functional deficiencies, such as spastic paralysis, does not alter the need for such institutional care.

MENTALLY DEFICIENT CHILDREN WITH PORENCEPHALY OR HYDROCEPHALUS EX VACUO OR BOTUL. After a pneumoencephalogram has revealed such permanent loss of brain substance as is incompatible with normal function, and after psychiatric examination has shown that the child is mentally defective, he should be treated in the same way and for the same reasons that are

lated under *Mentally Deficient Children with Normal-Sized Brains*. If such patients have other functional deficiencies, such as a spastic paralysis, this will not alter the need for institutional care.

MENTALLY NORMAL CHILDREN WITH SPASTIC PARALYSIS OR OTHER EVIDENCE OF LOCAL LOSS OF CEREBRAL SUBSTANCE Mentally normal children may show the results of earlier neglected craniocerebral birth injuries in a variety of ways. Depending on the location in the cerebrum of the cell destruction there may be hemianopias, spastic hemiplegias, spastic hemiparesis, monoplegia or diplegia, various types of tremor, athetoid movements, cranial-nerve palsies and so forth, all, however, accompanied by a normal or almost normal mentality. If the loss of substance of the cerebrum is larger the prognosis is obviously less hopeful than if the destruction is of the cellular order. The nature of and time devoted to such training as will provide the nearest approach to normalcy for the afflicted patient will of necessity be conditioned by this knowledge. It is essential, therefore, that a pneumoencephalogram be made before therapeutic gymnastics and other forms of exercise are begun. If this demonstrates porencephaly or a hydrocephalus *ex vacuo* the location and extent of the tissue loss, along with the necessary permanent functional loss, must be reported to the physiotherapist in charge of the rehabilitatory exercises. In addition, these patients should be referred for other training to persons especially trained in dealing with the deficiency. This may necessitate placing the child for a long time, either wholly or partly in a school for crippled children or may necessitate certain surgical measures of a corrective nature on the periphery but should not be taken as implying that such children should be permanently institutionalized. On the contrary, particular care must be taken to see that these children have the type of family understanding, sympathy and care that makes their home the one place they prefer to live in. This implies, of course, that within his physical limitations the deformed child accepts his full share of family responsibility, abides by family discipline and occupies no more important place in the family circle than any other comparable member.

THE TREATMENT OF INJURIES TO THE SPINAL CORD AND CAUDA EQUINA

All these patients must be hospitalized for therapy.

General Considerations

Proper care of a patient who has suffered an injury to the spinal cord or cauda equina will require the co-operative services of neurologic, genito-urinary, plastic and orthopedic surgeons, a dietician, a physiotherapist, a brace maker and a social-service worker. One of the first four of this group must assume control of and responsibility for the over-all care, the first choice being the member who is most enthusiastic, is willing to give the necessary time, has the best general knowledge of the problems involved and is willing to watch constantly the small details of therapy. Even these attributes are useless unless this surgeon is also convinced that no matter

DIAGNOSIS	PATHOLOGY	COURTESY- STENT TEST	INTRACRANIAL PRESSURE	CEREBRO- SPINAL FLUID	DIAGNOSIS MADE BY	SYMPTOMS	SIGNS	TREATMENT
Anatomic Transection	Complete anatomic severance of the cord or complete destruc- tion of the injured area, with or without a cyst of the lepto- meninges; no trans- mission of electrical impulses through the site of injury	Positive or negative	Normal or low	Clear or bloody Protein normal un- less C.S.F. is bloody	Operative vi- sualization of the injured cord with ver- ticalization by electrical stim- ulation above and below the point of injury	Loss of all voluntary motion and sensa- tion below level of injury; reflex bladder and bowel impar- tion; vaso-vascular reflex usually involuntary flexor-adductor spasms of legs ("mass reflex"), var- iable loss of sexual function; visceral pains; irregular sweating in the paralyzed area	"Mass reflex" hy- peractive tendon reflexes Babinski reflex muscular atrophy; prelec- sion to pressure and bed sores; res- piratory dehi- cally hypopro- teinemia anemia osteoporosis	Anterior rhizotomy elimination of fixed splints, especially plus- ter of pelvis; peripheral tendon and nerve op- erations two-hourly moving maintenance of dry bed; enemas high protein (150 gm. per 4 hrs.) diet high calorie (3000 cal. per 4 hrs.) diet 4800 cc. of broths per 4 hrs. tidal drainage bed cast cues sponges rubber mattress no Bradford or other frames
Physiologic transection	Incomplete anatomic severance of the cord or incomplete destruc- tion of the injured area, with or without formation of a syrinx and without any func- tion below the injured area; minimal trans- mission of electrical impulses through the site of injury	Positive or negative	Normal, high or low	Clear or bloody Protein normal un- less C.S.F. is bloody	As above	No "mass reflex," otherwise those of anatomic transection	Normal tendon re- flexes, otherwise those of anatomic transection	Anterior rhizotomy not necessary other- wise as in anatomic transection
Atrophy	Atrophy and shrink- age of the cord below the level of injury and interference with the blood supply; min- imal transmission of electrical impulses through the site of injury	Positive or negative	Normal	Clear Protein is normal	By visualiza- tion of the cord at laminectomy	Those of physiologic transection	Those of physio- logic transection	That of physiologic transection

TABLE 5 PARTIAL NONTRANSFECTING INJURY OF THE SPINAL CORD

DIAGNOSIS	PATHOLOGY	QUICKEN- TEST	INTRA- CRANIAL PRESSURE	CEREBRO- SPINAL FLUID	DIAGNOSIS MADE BY	SYMPTOMS	SIGNS	TREATMENT
Hematomyelia	Multiple macroscopic or small hemorrhages throughout the injured area	Negative	Usually normal	Clear or bloody Protein normal unless C.S.F. recovery from bloody	Quickened test and the signs and the symptoms present after recovery from spinal shock	Complete—changing gradually to partial—loss of all voluntary motion and sensation below the level of injury, no normal bladder and bowel normal skin—vascular reflex, occasionally adrenergic—flaccid and/or extensor spasms of arms and legs usually a flaccid paralysis of varying degree variable loss—usually none—of normal function	Occasional "mass reflex" of either flexor or extensor type normal tendon reflexes if no "mass reflex" Babinski reflex present or absent normal tendency to perspire and bed-sores; temporary respiratory difficulty anuria	Differential rhizotomy when indicated faced spinals with care and in moderate —bony movement maintenance of dry bed excreta high protein (150 gm. per 24 hrs.) diet high caloric (3000 cal. per 24 hrs.) diet 4000 cc. of fluids per 24 hrs. tidal drainage, only while the bladder is abnormal bed cleansed as soon as possible sponges rubber mattress no Bradford or other frame
Edema	Local spinal cord swelling and congestion, with or without hematomyelia and its pathology	Possible for a time	Usually normal	Clear Protein may be increased	The same method as in hematomyelia			
Constriction	Swelling, congestion, hematomyelia, intramedullary thromboses and hemorrhages, necrosis and liquefaction of spinal cord tissue	Negative or positive	Increased	Bloody Protein is increased	The same method as in hematomyelia, supplemented by laminectomy			
Isolated intramedullary hemorrhage	A local collection of blood clot in the substance of the spinal cord	Positive	Normal	Clear Protein is increased	Inspection and if necessary needling the spinal cord at a laminectomy	As in hematomyelia, edema and constriction	As in hematomyelia, edema and constriction	As in hematomyelia, edema and constriction, with laminectomy and incision of spinal cord and traction of clot, or drainage of syrinx by needling or incision with suturing of cut edges so as to hold the cavity open
Traumatic syringomyelia	Syringomyelia cavity in the spinal cord	Positive	Normal	Clear Protein is increased	Inspection and needling at laminectomy			

TABLE 6. SPINAL CORD AND CAUDA EQUINA INJURIES IN GENERAL

DIAGNOSIS	PATHOLOGY	OURCE- STIFF TEST	INTRA- CRANIAL PRESSURE	CEREBRO- SPINAL FLUID	DIAGNOSIS MADE BY	SYMPTOMS	SIGNS	TREATMENT
Spinal shock	Unknown Reacts with exanthema, hypoproteinemia, infection, anemia	Positive or negative	Normal	Clear or bloody Protein normal or increased	History of a significant spinal cord injury	Those of the spinal cord injury	Disorganization of all somatic and visceral reflexes, paralytic ileus, stomal block, tendency to bed sores	Total drainage 2-hourly turning Ochsnerian
With compression	Swelling of cord Closure of subarachnoid space	Positive	Normal or increased	Clear or bloody	Quickens test with or without x-ray confirmation	Those of the spinal cord or cauda equina injury	Those of the spinal cord or cauda equina injury	Keep bed dry Rectal tube No laparotomy
Without compression	Transsection of cord Partial cord lesion Atrophy of cord Disorganization of cauda	Negative	Normal	Increased protein Clear or bloody Normal protein	Quickens test with or without x-ray confirmation	Those of the spinal cord or cauda equina injury	Those of the spinal cord or cauda equina injury	Decompression by either lysterization of spine or laminectomy or both
Concussion	Caused by gunshot wound only and without contact between bullet and cord or meninges Pathology may be either that of transection or a partial cord injury	Positive or negative	Normal	Clear or bloody Increased or normal protein	By history with x-ray confirmation of bullet wound of bony canal	Those of the spinal cord injury	Those of the spinal cord or cauda equina injury	No treatment other than that necessary for the associated cord or cauda equina injury

TABLE 7 CAUDA EQUINA INJURIES

DIAGNOSIS	PATHOLOGY	CURRENT FINDINGS	INTRACRANIAL PRESSURE	CEREBRO-SPINAL FLUID	DIAGNOSIS MADE BY	SYMPTOMS	SIGNS	TREATMENT
Total Lacerations	Division of II of the nerve of the cauda equina below the tip of the cauda	Positive or negative	Normal	Clear or bloody Protein normal	Laminectomy	Flaccid peripheral- nerve type of sensory and motor paralysis below the level of 1st lumbar dermatome reflexes absent retrograde bladder incontinence	No "mass reflex" Normal or dimin- ished or absent tendon reflexes Muscular atrophy No Babinski re- flex, pressure but no bed sores ab- sent, normal treflex- gram flaccid sphincters; total urinary incontinence variable loss of sex ual function radicu- lar or vesicospastic types of pain	Repeated (every 6 hrs.) lumbar punctures with withdrawal of bloody cerebrospinal fluid de- compressive laminec- tomy lysis of roots caught in scar" driv- en" after stimulation and if inactive, of roots that cannot be freed and that are com- pressed or irritated diminution of fluid spinalis, especially plas- tic to back in o-phory moving malposition of dry bed removal high protein (1.50 gm. per 24 hrs.) diet high caloric (5000 cal. per 24 hrs.) diet tidal dial- ysis hyperextension by blanket roll if neces- sary bed exercises early mobilization as indicated
Total Destruction	Greatest wound with destruction and lacer- ation of the cauda equina, compression and destruction of cauda by scar from organized blood asso- ciated with a major fracture of a lumbar vertebra	Usually positive negative during the stages of from blood in the C.S.F.	Normal	Clear or bloody Protein is increased	Laminectomy supplemented by x-rays and lumbar punc- ture data	Isolated peripheral- nerve type of paral- ysis of varying degree and distribution pain only rarely otherwise as above	No "mass reflex" No Babinski re- flex, pressure but no bed sores ab- sent, normal treflex- gram flaccid sphincters; total urinary incontinence variable loss of sex ual function radicu- lar or vesicospastic types of pain	As above except that no decompressive laminectomy is neces- sary
Compress- ion	Compression of part of the cauda equina by scars or intradural pressure and adhe- sions originating as blood in the C.S.F., from bone fragments, etc.	Positive	Normal	Clear or bloody Protein is increased	Laminectomy supplemented by x-rays, tom- o-gram puncture data and Queckenstedt test	Isolated peripheral- nerve type of paral- ysis of varying degree and distribution pain only rarely otherwise as above	No "mass reflex" Irregular reflex re- sponses in accord- ance with amount of destruction of the cauda; reflexes, autonomous or not normal bladder; normal or uncontrolled bowel otherwise as above	As above except that no decompressive laminectomy is neces- sary
Partial division	Division of part of the cauda equina by frag- ments of bone, for e.g. bones, etc.	Negative	Normal	Clear Protein is normal	X-rays, lumbar puncture data, Queckenstedt test clinical symptoms and signs	Isolated peripheral- nerve type of paral- ysis of varying degree and distribution pain only rarely otherwise as above	As above	As above except that no decompressive laminectomy is neces- sary

TABLE 8. SPINAL CORD AND CAUDA EQUINA INJURIES: THERAPY ACCORDING TO LEVEL OF INJURY

LEVEL OF INJURY	RESPIRATION	SKIN	BLADDER	BOWEL	URINE	ILLNESS	OPERATION	THERAPY OF SPINAL INJURY	EXERCISE	AMPUTATION
Cervical	Early	Chest strap Oxygen Occlusive Empty the bladder	2-hourly turning Dry bed No fixed splints No plaster Plastic on bed sores	Tidal drainage	Edema	High protein, high calorie	4800 cc. per 24 hours	Decompression if necessary after 7 to 10 days	None	None
	Late	None	None	Bladder training	Bowel training	High protein, high calorie	4800 cc. per 24 hours	Dorsolumbar anterior rhizotomy if indicated	Bed and mast	Yes, with splints such as splints for par- alyzed hands
Dorsolumbar	Early	Protect tho- racic area as above	As above	Tidal drainage	Edema	As above	As above	Decompression if necessary	Hyperextension with bladder roll, no plaster	None
	Late	None	As above	As above	As above	As above	As above	Decompression if necessary	Hyperextension with bladder roll, no plaster	None
Sacral cord	Early	Protect tho- racic area as above	As above	Tidal drainage	Edema	As above	As above	Dorsolumbar anterior rhizotomy if indicated	Mistal Taylor back brace, collar, etc., no plaster	Yes, full course
	Late	None	As above	Tidal drainage	Edema	As above	As above	Decompression if necessary	Hyperextension with bladder roll, no plaster	None
Cauda equina	Early	None	As above	Tidal drainage	Edema	As above	As above	None	Mistal Taylor back brace, collar, etc., no plaster	Yes, full course
	Late	None	As above	Tidal drainage only if necessary	Bowel training if necessary	Normal	As above	Decompression if necessary on account of fall time of lumbar prosthesis drainage	Hyperextension with bladder roll no plaster	None
								Decompression of cauda, cor- dectomy etc.	Mistal Taylor back brace as indicated	Yes, full course

how extensive the paralysis may be in such a patient and provided only that he has full use of his hands, arms and shoulders, ambulation with infallible twenty four hour control of bladder and bowel (without the need of a urinal or other artificial aid)—as well as that degree of over-all rehabilitation that comes only with an ability to lead a normal social and work life within the limits imposed by the necessary use of braces and crutches—is well within the possibilities of present-day treatment. Failure to attain these ends is excusable only on the grounds of uncooperation by the patient and because of such other major organic damage as to make them physically impossible of attainment.

Diagnosis

Spinal-cord injuries fall into twelve diagnostic classifications: spinal shock, transecting and partial cord injuries, transecting, totally and partially destructive cauda-equina injuries, compressive and noncompressive injuries of both cord and cauda, concussion of the cord, atrophy and traumatic syringomyelia of the cord, and radicular injuries. *Spinal shock* accompanies every significant cord injury. It causes chaotic areflexia of both the somatic and visceral varieties. It is not present in cauda injuries. Because of this characteristic, atonicity of the bladder and bowel and loss of the skin vascular reflex are present as long as it is present. It recurs with exhaustion, anemia, hypoproteïnemia, infection and after major surgery. *Concussion of the cord* is associated only with gunshot wounds that are limited to the bony wall of the spinal canal. It may be either a transecting or a partial injury. *Atrophy of the cord* is presumably the result of vascular inadequacy. It commonly produces symptoms of transection, but not necessarily so. *Traumatic syringomyelia* is usually the end result of intramyelogenous necrosis. The cavity is limited in extent to the approximate area of injury. It commonly produces symptoms of only a partial lesion. *Transections* show loss of sensation and voluntary motor power, either with or without associated adductor-flexor-extensor spasms of the muscles of the arms, abdomen, spine and legs in accordance with the level of the injury. If the cord is involved, the same signs and symptoms apply to the cauda injuries, except that spasms never occur. *Partial lesions* of both cord and cauda leave varying degrees of sensation or voluntary motion or both in the area below the level of injury. *Partial cord injuries* may or may not cause adductor-flexor-extensor spasms of the nonparalyzed muscles. *Destructive cauda injuries* cause symptoms that are analogous to transections of the cauda but are more extensive. *Both compressive and noncompressive injuries* may be associated with either a transection or a partial injury of the cord and cauda or with a totally destructive lesion of the cauda. *Compressive injuries* show a complete or partial cerebrospinal-fluid block by the Queckenstedt test and by an increase of the protein content of cerebrospinal fluid taken from below the block. *Radicular injuries* show signs of irritation, compression or interruption of individual roots and their peripheral fields of supply.

Symptom Complexes

From the point of view of symptom complexes and in addition to the characteristic sensory and voluntary motor loss, the injuries fall into six groups. They are those of the cervical segments the upper two-thirds of the thoracic cord the lower one-third of the thoracic and upper three fifths of the lumbar cord the lower two-fifths of the lumbar with all the sacral cord the conus and the first inch of the cauda the cauda below the level of the conus and the radicular injuries. The *diagnostic characteristics* are cervical—respiratory deficit and anoxia upper thoracic—respiratory deficit, associated pulmonary and mediastinal symptoms and a high incidence of bed sores lower thoracic and upper lumbar cord—a high incidence of bed sores as well as liability to sexual impotence and sterility in the male in cases with very extensive destruction of cord tissue lumbosacral cord conus and upper one inch of cauda—denervation of the bladder lower bowel and their sphincters as well as liability to sexual impotence and sterility in the male cauda equina and radicular injuries—motor and sensory deficits characteristic of cervical thoracic lumbar and sacral root or peripheral nerve injuries and corresponding to irritation compression or interruption of the involved root or roots.

The Associated Bone Injury

This is diagnosed by x ray examination. It may involve any part of the bony intervertebral disc, cartilaginous, or ligamentous structures that make up the vertebral column. *Fractures* may occur alone or in conjunction with anterior lateral and (rarely) posterior dislocations either with or without extrusion of a nucleus pulposus. *Dislocations* do not occur alone. If no fracture is visible by x-ray extensive ligamentous injury with or without extrusion of a nucleus pulposus must be presumed. *Subluxation*—that is, dislocation with spontaneous reduction—is not uncommon. It is usually associated with ligamentous damage or with such damage in conjunction with minor fractures often invisible by x ray examination. *Major fractures of the twelfth thoracic vertebra and of all the lumbar vertebrae* commonly bleed into the lumbar subarachnoid space. The organization of the blood by the end of fourteen to twenty-one days may compress the cauda and initiate neurologic symptoms then for the first time.

Protrusion or Extrusion of Nuclei Pulposi

Protrusion or extrusion of all or part of the nuclei pulposi of any of the intervertebral discs through a ruptured annulus may cause compression and angulation of the cord or irritation, paresis or paralysis of one or more roots of the cauda or cervical cord. This occurs most commonly in the low lumbar area. The next most common sites are the lower cervical and upper lumbar but the lesion may occur at any level.

Transportation

All patients with a cord or cauda equina injury of whatever degree and all patients who exhibit any peripheral voluntary paralysis, no matter how

fleeing, at the time of the injury *must* be transported *face down* on a solid litter or some substitute that is longer than the patient and wide enough so that he will not roll off. The victims must be *rolled* and *never* lifted onto this litter and not moved at all at or from the scene of the accident, until such equipment is available except for such amount as may be necessary to assure adequate respiration or control of major hemorrhage. *Cervical cases*—that is, any patients with any degree of paralysis of all four extremities—should have a roll of folded newspaper or a folded turkish towel wrapped around the neck and held in place with string or adhesive plaster even before they are turned, and especially before they are removed from any vehicle that they may be in. *Under no circumstances* should flexion of the head forward toward the chest be permitted for any purpose whatsoever. Hot water bottles or other heaters must be used with the greatest care to avoid burning any anesthetic or insensitive areas.

If the patient is in *severe shock* he should be transfused with whole blood at the site of the accident and before being moved.

Procedure on Admission to the Hospital

If the patient's condition permits, he should be taken to the ward by way of the x ray room, where enough films of the spine should be taken to give some idea of the bone damage, as well as a single film of the lungs. The chest film should be anteroposterior and designed to show any air or fluid in the pleural cavity or pathology in the lungs themselves. The spine films should be anteroposterior and lateral views and (if possible, and if the patient's condition permits) should be stereoscopic, otherwise the taking of x rays should be postponed until later. On admission to the hospital the ward should be notified, and the bed should be made up with a draw sheet over a sponge rubber mattress on top of a hair mattress and spring.

If the patient is in severe surgical shock on admission, 500 cc. of properly matched whole blood should be administered intravenously in the emergency or accident room before he is moved further.

He should be moved only on a full-length, sufficiently wide litter in the face-down position *always rolled* and *never* lifted, and under no circumstances should his head be flexed on his chest until after the level and extent of the cord injury is determined.

After the patient has been admitted to the ward and *rolled* off the litter onto the already prepared bed, the following procedures should be carried out in approximately the prescribed order in *all patients with cord and cauda injuries*.

- 1 The systolic and diastolic blood pressures should be taken. If the pulse pressure is 20 or lower or if the systolic pressure is below 100 in an adult, or if the pressure is within normal limits and there is clinical evidence of surgical shock (such as cold extremities, pallor, sweating, restlessness, apprehension and so forth) 500 cc. of properly matched whole blood should be given intravenously together with one ampoule (7½ grains) of caffeine and 1½ grain of strychnine, also intravenously. Heaters and blan-

THE CENTRAL NERVOUS SYSTEM

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kets should be used as indicated but with care to avoid burning any anesthetic area. No further procedures except catheterization as described below should be carried out until the patient is out of surgical shock except to repeat the above in two hours or less if necessary.

2 Fixation of the bone injury in extension—with traction if necessary—should be assured.

3 A No 14 F whistle tipped sterile catheter or No 14 F sterile rectal tube should be inserted into the bladder and held in place by appropriate three-tailed adhesive strapping to the penis. If the patient is a woman, the catheter should be a No 14 mushroom-tipped sterile catheter held in place by adhesive strapping to the pubis or thigh or both. Smaller catheters should be used for children. Except when specifically indicated, Foley type catheters should not be used in either sex. A catheter or rectal tube larger than a No. 14 F should never be used in these cases and the catheter must be of the type that has the opening in the bladder-end in the end and not on the side of the tip.

The bladder is now emptied. If it is estimated that it contains 100 cc. or more of urine or if it is distended or even overfilled no more than 25 cc. of urine should be allowed to drain out at any one time. Under such circumstances the intervals between such drainages must be no less than five minutes by the clock. After the bladder has been emptied the open end of the catheter is covered with a sterile piece of gauze held in place by an elastic band.

4 If the patient is not in surgical shock or has recovered from shock by virtue of the therapy provided, a brief physical and neurologic examination is now performed. The purpose of this examination is to determine roughly the condition of the patient's heart, lungs and abdomen (including audible peristalsis) the presence of any other significant injuries as well as approximate upper level of any loss of sensation, any loss of voluntary motion and the activity of the anal reflex. The less examining done at the time—provided such desired information is obtained—the better.

5 Portable x-ray films of the spine should now be taken at the level indicated by the neurologic examination, as well as films of any other bony injuries, and a film should be made of the chest to show the condition of the lungs and pleural cavity if this has not already been done. The films should be examined while still wet, and the doctor in charge of the patient notified at once of any pathology seen.

6 While waiting for the report from the radiologist a specimen of urine should be collected and sent for examination. Enough blood should also be collected for serology serum protein, nonprotein nitrogen, sugar carbon dioxide combining power and blood typing. A complete blood examination should be made, including red count, hemoglobin, white count, smear and hematocrit.

7 If the patient is unable to urinate voluntarily preparation should now be started to put him on tidal drainage (see page 84)

8 The following orders should be written for the nursing staff

Record pulse temperature respiration and blood pressure every four hours and oftener if ordered or indicated.

Measure and record fluid intake and output.

Stop all food and fluids by mouth, including water and cracked ice, until further orders.

Change the patient's position from side to back and to the other side every two hours day and night. Change of position should be accomplished by lifting and turning of the patient on and with the draw sheet while his head is rotated by an assistant (see page 128)

Change the patient's bed and clothing, clean dry and powder soiled or wet skin within fifteen minutes of the time the bed is either soiled or wet.

Rub the patient's back hips and heels paying particular attention to the sacral and trochanteric areas, every four hours until otherwise ordered.

Administer no drugs unless specifically ordered by a doctor

Report significant changes in blood pressure, pulse rate respiratory rate and temperature Report changes in color of face feet or hands. Report drowsiness unconsciousness, convulsions, unco-operation, confusion and mania. Report redness or any break in the skin of the back, hips, buttocks, heels, knees, chin and occiput.

Adjust paralyzed limbs in midflexion with supports for the feet. Protect the patient from the damaging effects of local pressure from weight bearing by free use of pillows under bony supporting points.

Do not use doughnuts rubber rings, Bradford or other type of frame.

Give an enema daily unless otherwise ordered.

Report suspected fecal impaction at once.

9 Traction or hyperextension or both—according to the level of the cord injury as described below under the appropriate heading—should now be instituted.

10 A lumbar puncture should be done with a manometer on the needle. After abdominal compression to assure the patency of the needle and manometer the presence or absence of a cerebrospinal fluid block should be determined by means of a fractionated Queckenstedt test (see page 239) Five centimeters of cerebrospinal fluid should then be withdrawn, enough used for a red and white cell count and the rest spun down for a total protein determination on the centrifuged fluid.

11 If the abdomen is silent or distended a rectal tube should be inserted and left in place for thirty minutes out of every hour Vomiting or upper abdominal distention should be treated by Wangensteen suction which should be started at this time.

12. Intravenous fluids in amounts sufficient to provide 4500 cc. per twenty four hours for an otherwise normal adult should be started. Ten per cent glucose alternately in distilled water and isotonic salt solution should be used intravenously

13 The tidal drainage (see page 84) should be set up and the urethral catheter attached to it.

14 A cystometrogram (see page 89) should be made.

THE CENTRAL NERVOUS SYSTEM

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15 Arrangements should be completed to administer 150 gm of protein intravenously every twenty four hours by the use of an amino acid solution while the abdomen remains silent or distended and during the period that food and fluids are prohibited by mouth.

Treatment

Patients with spinal cord and cauda equina injuries fall into six therapeutic groups or classes.

Class 1 The Stage of Surgical and Spinal Shock

SURGICAL SHOCK. This must be treated more vigorously than usual. Repeated whole-blood transfusions are the best method. If hot applications are applied extra care to avoid burning the anesthetic areas must be taken.

RESPIRATORY DIFFICULTY. In Cervical Cases The airway must be kept clear. The use of respirators of any type or of masks as a means of supplying oxygen is usually impossible and unless accepted cooperatively by the patient are contraindicated. Oxygen must be administered constantly however until the respiratory exchange is balanced and fixed—a matter of days or weeks. This is best done through an inlying nasal catheter. The chest wall must be immobilized by wide adhesive strapping made by placing two three-inch strips together sticky side to sticky side in such a way that there will be an overlap of three inches at one end. The combined strap is applied as tightly as possible around the chest in expiration, the adhesive overlap being stuck to the nonadhesive surface of the strap and anchored there by a safety pin. If there are pharyngeal, tracheal or bronchial secretions of any moment it is well to liquefy them as much as possible so that they may be more easily coughed up. The use of steam inhalations (without any adjuvant) and postural drainage, in so far as it is compatible with the traction in extension that is mandatory will often prove extremely useful. Paralyzed arms must be kept off the chest and abdomen. In Thoracic Cases A thoracentesis must be performed at any suspicion of tension pneumothorax, hemopneumothorax, fractured ribs or pneumonia, and especially when pneumonia has been diagnosed because this diagnosis is almost certain to be wrong. If fluid or intrapleural air under tension is found it should be treated appropriately the thoracentesis being repeated often enough to empty the pleural cavity and prevent reaccumulation. Morphine should be given in doses large enough to control any major pain but not large enough to depress the respiratory center in severe injuries. In these less severe instances where the pain is traceable to root irritation or compression the danger of an unexpected respiratory paralytic effect is less. The drug is usually unnecessary after fixation of the bone. Respiratory difficulties with injuries at levels other than the cervical or thoracic present no special problems except when complicated by fractured ribs or a hemopneumothorax. In the former the chest should be strapped as described under Cervical Injuries above. The hemopneumothorax must be treated by repeated thoracenteses.

ABDOMINAL DISTENTION *All Levels* This should never be allowed to develop. All foods and fluids by mouth, including water and cracked ice, must be stopped for as long as the abdomen is silent and especially if it is distended. A rectal tube should be kept in place for thirty minutes out of every hour. The stomach must be kept empty with Wangensteen suction, and depressant drugs such as morphine and codeine should be avoided. Other drugs, such as prosligmine, that are bowel stimulants are also contraindicated at this stage as are hot applications, turpentine stupes, enemas and the like. Laparotomy no matter for what diagnostic reason, is contraindicated unless the surgeon is willing to run the risk of being unable to replace the distended paralyzed loops of bowel in the peritoneal cavity in the case of a negative exploration done under the mistaken diagnosis of an intra-abdominal hemorrhage or a ruptured vessel. After peristalsis is again audible food and fluids may be started by mouth.

THE BOWEL. After distention has disappeared and peristalsis is re-established the bowel should be cared for by daily enemas, preferably given at the same time every day in order to thus initiate the "bowel training" program (see page 126). Care must be taken to prevent the development of fecal impactions which, when they are present, must be removed manually. Mineral oil given by mouth in appropriate dosage is helpful. The usual type of enema will be soapsuds or oil but at times a milk-and-molasses one will prove helpful and necessary. Cathartics are not necessary.

THE BLADDER. The bladder should be treated with tidal drainage (see page 84) with the siphon loop set at 5 cm. if the cystometrogram (see page 153) shows an atonic bladder. Appropriate adjustments should be made in the height of the siphon loop for other stages of bladder recovery. A cystometrogram should be made at least once a week unless the bladder is normal, in which case it needs no treatment. No other method of treatment is satisfactory. In particular emptying by overflow incontinence by manual expression of the bladder contents or through a suprapubic or perineal cystostomy are all contraindicated. The catheter must be either whistle-tipped or a rectal tube and must under no circumstances exceed No. 14 F in size. Foley type catheters should not be used except for special indications.

THE BONE INJURY. The bone injury should be treated by *slow* hyperextension with traction. It is theoretically possible that in cervical cases there may be such an injury present as to further damage the cord by extension of the cervical spine if it is effective. Such an injury requires traction in a straight line and without hyperextension. I have seen this only in patients with locked cervical facets in which the hyperextension had no effect until the associated traction had unlocked the facets, and I doubt if the theoretical danger of further cord damage is enough to counterbalance the practical good of prompt hyperextension if it is slow and accompanied by traction.

The Cervical Cases. Traction and hyperextension can be carried out as an emergency measure by hanging 5 pounds to one end of a line which is carried over a pulley and has the other end attached to a spreader over

which are looped the ends of a home made bridle constructed of folded flannel bandages placed under the chin and occiput respectively and pinned opposite the ears (Fig. 7) For permanent traction which should be established as soon as possible more weight up to 30 pounds may be used with the help of Crutchfield-type tongs (Fig. 8) (see page 235) These may be applied if necessary without moving the patient from his bed. Traction should be maintained for at least six weeks If the injury has been



Fig. 7 Flannel bandage halter for traction and extension of the cervical spine in the emergency treatment of a cervical cord injury Note the intranasal administration of oxygen.

ligamentous the traction should be kept on for eight to twelve weeks. After the traction has been removed the patient must wear a Zummer type splint with a pelvic attachment (see Fig. 29 page 136) until all danger of secondary collapse or redislocation is past. Plaster of paris splints or casts for treatment of any associated bone injury are specifically contraindicated at all times in cervical cord injury cases. If used they produce a great diminution in respiratory efficiency and may cause a death that would not occur otherwise These deaths are commonly diagnosed as being due to either pulmonary emboli or pneumonia. Fusion whether by bone inlays or by

wiring of the spinous processes either alone or in combination, is contra indicated at this time. Closed manual reduction with or without an anesthetic should never be attempted.

Other Levels. Hyperextension is best carried out by placing boards between the mattresses and bedspring with a rolled blanket between the boards and the mattresses opposite the kyphos (Fig. 9). By properly adjusting the



Fig. 8. Crutchfield type tongs for traction and extension of the cervical spine in the definitive treatment of a cervical cord injury.

slant of the bed, traction and counter traction will be maintained by the weight of the upper body on one side and that of the pelvis and legs on the other. Further artificial traction is not necessary. The rolled blanket should be added to every day or so until the proper amount of hyperextension has been obtained. These patients must be kept in bed in hyperextension for six to eight weeks or longer depending on the degree of recovery of normal bone contour and solid weight bearing repair that has taken place. This will be determined by the x ray findings. They should then be fitted to an Armstrong type of spring-steel or other standard back brace until x ray evidence of a bony union of the injured vertebrae is unquestionable. The brace must be worn whether they sit or stand. Violent exercise within the

brace is contraindicated but muscle setting exercises and mild bed exercises may be practiced. Plaster of paris casts are contraindicated at all times in all cord injuries and in all *cauda equina* injuries because of the resultant high incidence of bed sores and the prolongation of convalescence with other wise unnecessary surgery and so forth that follows.

MOVING All patients with an injury to the spinal cord and cauda equina must be turned every two hours day and night as long as they are in bed. They should be turned with the aid of the draw sheet (see page 128) All Bradford or other such frames or devices previously advocated for moving these patients are either contraindicated or unnecessary.



Fig. 9 The use of a blanket roll placed between mattress and bed-boards as a method of treating fractures of the thoracic and lumbar spine by hyperextension.

NURSING The bed should be made with the usual sheets and blankets, including a rubber and a linen draw sheet. A hair mattress should be used underneath a foam-rubber mattress. Air and water mattresses are contraindicated. Sectional, split and "mobile mattresses" are not necessary. The patient's back must be kept dry clean and powdered constantly and massaged at least twice a day and every four hours during the early stages. Fifteen minutes in a wet bed has only too often produced a bed sore that has added many unnecessary months to the patient's convalescence and hospital stay. Nursing discipline must be maintained. Emotional storms and exhibitions of temper on the part of the patient are to be anticipated and should be treated as they would be in a small child. Deliberate obscenities and unco-operation, however, should not be countenanced. If they do occur the nurse should stop whatever she is doing, and leave the patient exactly as he happened to be at the moment and not return or render any further service to him unless and until such time as he indicates his appreciation of his actions and his intention not to repeat them. Nurses cannot be asked or expected to care for those patients who repeatedly transgress the rules of common decency.

DIET As soon as possible these patients should be given a diet that contains 3000 calories and 150 gm of protein per day. Great care should be taken to make sure that it is ingested. A favorable nitrogen balance *must* be maintained. Amino acids in commercial preparations, as well as glucose and salt, can be given intravenously if necessary during the early stages. Later tube feeding can be resorted to if peroral feeding is impossible. It is preferable to give the diet by mouth as soon as possible however. It is probable that many of these patients suffer from some degree of liver dysfunction. It is not known at present how to prevent or treat this. Vomiting and gastro-intestinal upsets which sometimes are associated with the ingestion of such a high protein diet can usually be prevented by reducing the fat content to a minimum. All vitamins must be administered in large doses. This diet should be started at once after the accident and must be continued until the patient is up and about, eating a normal diet and able to get out of doors.

FLUIDS The fluid intake must not be allowed to fall below 4500 cc per twenty four hours for an otherwise normal adult. This should be scaled down to appropriate levels in the case of children and cardiac and renal invalids.

CEREBROSPINAL FLUID During this stage careful watch should be kept on the protein content of the cerebrospinal fluid. A rise may be the earliest sign of a beginning block and enable the doctor to recognize a compressive lesion in its early stages. Samples of cerebrospinal fluid are collected by lumbar puncture. The collection should always be preceded by a study of the dynamics by means of a fractional Queckenstedt test (see page 239). Compression of the jugular veins in this test should not be done digitally but rather as a serial compression by the inflation of a sphygmomanometer cuff which has been wrapped around the patient's neck. The observations are made every 10 cm from 0 to 40 and reverse. Gross blood in the cerebrospinal fluid with a major fracture of a lumbar vertebra necessitates daily lumbar drainage of all the cerebrospinal fluid. It is practical to remove at each puncture. This should be continued until the cerebrospinal fluid is clear and colorless and its protein again normal.

PRESSURE AND BED SORES Pressure sores should not occur in any patient and will not if the patient is turned every two hours, his back kept clean and dry, his nitrogen balance and serum protein maintained at normal and all splints or supports that are fixed in relation to his bony weight-bearing points done away with. Bed sores or decubiti never develop except after pressure sores and in connection with maceration of the horny layer of the skin, or by subcutaneous ischemic necrosis. When decubiti are present the surrounding skin should be kept covered with tincture of benzoin (unless the patient is allergic to it), necrotic tissue removed surgically and a dry sterile dressing applied to the denuded ulcer. If the bed sore is large and there is a significant loss of serum and its contained protein the area should be skin-grafted as soon as possible as a temporary measure. This may prove to be a life saving procedure. Surgical repair should not be undertaken

during this stage Meticulous care must be given especially to thoracic and upper lumbar cord injuries because they are particularly liable to develop complicating bed and pressure sores.

OPERATIONS. All major surgical procedures are contraindicated during this stage of surgical and spinal shock and for as long as is safe and wise thereafter. In particular cervical laminectomies, even in the presence of compression of the cord, must, if at all possible, be postponed. This admittedly increases the amount of cord damage but it is preferable to the outrageously high mortality that accompanies such a procedure at this time. If a cervical operation cannot be postponed for adequate reasons the operation should be a hemilaminectomy the interspinous ligament left intact the surgery performed with the patient on his side rather than face down and traction preferably by way of Crutchfield-type tongs, maintained throughout. Provision for a decompression should be provided by leaving the dura open at the close of the operation (see pages 231 and 234). In general, cervical laminectomies should not be performed for seven to twelve days and thoracic not for five to seven days after injury. Lumbar laminectomies may be done if necessary twenty-four or forty-eight hours after injury depending on the patient's general condition. In particular no operation done for the purpose of openly reducing a dislocation can be countenanced in these patients with cord injuries.

Class 2 The Stage of Diagnostic and Reparative Surgery

Patients in this stage will have recovered from their surgical shock. They may or may not be still in spinal shock. As a prerequisite to any operative procedures the patient must be well nourished and gaining weight, in nitrogen balance and his nutrition must be "on the mend."

GUNSHOT WOUNDS, STAB WOUNDS, COMPOUND FRACTURES OF THE SPINAL CANAL, COMPRESSION OF THE SPINAL CORD OR CAUDA EQUINA OPERATIONS Débridement (see page 234) Whenever there is a traumatic communication through the skin to the deeper structures and especially to the bone or dura, a classical débridement of all contaminated tissue along the track with removal of all loose fragments of bone and foreign bodies should be carried out as early as possible but in any event within seventy-two hours of the time of injury if the patient is not in surgical shock. Chemotherapy and antibiotics must be used freely but they cannot be regarded as a substitute for a proper débridement. If the dura has been opened as a part of the injury its edges should be débrided and it can be left open. It should not be deliberately opened by the surgeon, however. Nothing should be done to the cord itself or to the elements of the cauda. Irrigation of the wound with salt solution or other fluid is contraindicated. Penicillin and sulfanilamide crystals should be left in the wound and especially intrathecally.

Decompression (see pages 231 and 234) The need for a more accurate diagnosis of any pathology that is present can now be met by a diagnostic laminectomy. At this operation the injured area of the cord, as well

as a normal segment or two above and below should be exposed and stimulated electrically below at the site and above the injury. This verifies the presence of a transection by the absence of muscular contractions in myomeres *below* the injury as the direct and immediate response to electrical stimulation of the anterior cord *above* the injury. Syringomyelic cysts should be incised and their edges sutured back to make certain that they stay open. Any cerebrospinal-fluid block that is present should be relieved by decompression, the dura in these instances being left open. In my experience open reduction of dislocations by forcible instrumental leverage of facets is too dangerous to be practiced in these cord injuries. An exposure that is too wide is preferable to one that is too small and enough laminae, regardless of the number should always be removed to insure this. The postoperative care of upper thoracic laminectomies should include limited fixation of the shoulders and upper arms to prevent tearing out of the stitches that are used to approximate the interscapular muscles, with partial separation of the wound.

Rhizotomy for Spasm. This should be an anterior rhizotomy and should be done only after visualization and electrical stimulation of the damaged area of the cord has made it certain that the injury there is actually a complete anatomic transection. The tenth, eleventh and twelfth thoracic, the five lumbar and first sacral anterior roots must be divided bilaterally and after electrical stimulation. The second sacral root is identified by electrical stimulation with resultant bladder contraction as evidenced by the production of a true emptying contraction in a cystometer attached to the partially filled bladder by way of an indwelling catheter. The second sacral root and the roots below it should never be divided. Rootlet rhizotomies for the relief of spasm associated with a partial cord injury should not be performed at this time. Posterior rhizotomy for spasm is often not effective at all and is always less effective than anterior. Rhizotomy by means of blind intrathecal injections of alcohol is inaccurate, ineffective and carries an unpredictable chance of permanently paralyzing the bladder and bowel and especially their sphincters (see also page 109).

Surgical Repair of Bed Sores. This should be carried out during this stage. Skin-grafting of the sore itself if resorted to must be regarded as a temporary procedure only. The choice between linear suture, rotation flaps, double pedicled flaps and delayed flaps will depend on the size of the ulcer, its location in relation to weight-bearing points, the patient's resistance and the surgeon's judgment. Z type repairs are not suitable. In any case all scar tissue must be excised, underlying bone removed sufficiently to do away with all osteomyelitis and to expose a cancellous bleeding surface, the walls of any burn that may be present removed and the flap sutured in many layers without tension. Hemostasis must be meticulous. Drainage should be provided, nevertheless and must be through a stab wound and not through the incision. The drainage material should be a fenestrated stiff-walled rubber tube. The type of suture material is of no consequence except that catgut is not suitable. There are other equally important

technical details with which the surgeon must familiarize himself before undertaking surgical repair of these lesions.

Renal and Bladder Operations Any necessary renal and bladder operations for acute conditions such as require the removal of stones and the closure of suprapubic and other bladder or urethral fistulas should be done in this stage. Pyelotomies, ureterostomies, resection of the urethral sphincter and injection of the sacral spinal nerves supplying the detrusor and sphincters (see pages 104 and 105) should not be done at this time however. Nephrectomies should be done only for specific indications and if they cannot be avoided.

Osteomyelitis of the Hip Joint Excision of septic osteomyelitic acetabula and heads and necks of femora can be done during this stage if indicated. Cup arthroplasties are contraindicated in these patients at all times.

THE BONE INJURY It is during this stage that the bone injury heals sufficiently to insure stabilization in the face of early moderate exercises and the start of weight-bearing. This second stage does not end until this has been accomplished. The use of plaster of paris and other splints that are fixed in relation to the patient's body must still be avoided.

THE BOWEL As in class 1

THE BLADDER The bladder is maintained on tidal drainage (see page 84). By the time the patient is ready to leave this stage his bladder should be reflex or normal providing it and its sphincters have not been denervated by the cord or cauda injury.

DIET As in class 1

PHYSIOTHERAPY If the paralysis is flaccid, physiotherapy should be started and pressed actively during this stage. Muscle setting, active assisted motion, passive motion, massage and maintenance of full range of motion in all joints should be instituted and maintained. Physiotherapy of the paralyzed extremities is contraindicated in the presence of "spasms" or hypertonic muscles.

OCCUPATIONAL THERAPY Short range diversional, easily accomplished projects should be started.

NURSING Except for quadriplegics all special nursing should be eliminated during this stage. Failure to do so prevents the development of that essential stimulus that comes only from self-care. For this reason the patient must be moved out of any private or semiprivate hospital accommodation he may have had up to this point and be placed in a ward instead.

BED EXERCISES The importance of bed exercises cannot be over-estimated. They are the foundation upon which all later physical rehabilitation rests. They should be started as soon as the bone injury permits. Their scope, severity and length should be increased as rapidly as possible. A back-splint may be used if necessary. Deep-breathing exercises, push ups from a prone position, work with dumbbells and spring exercisers to develop handgrip, upper arm, shoulder and scapular muscles and the setting of abdominal, back and leg muscles must all be pushed actively. The patient should start to learn to balance himself while sitting erect in bed and

to initiate forward flexion exercises at the waist. In this way he will mobilize his hip joints and thus learn to reach his feet. This permits him to put on his own shoes, socks and splints and thus facilitates adequate self-care and initiative

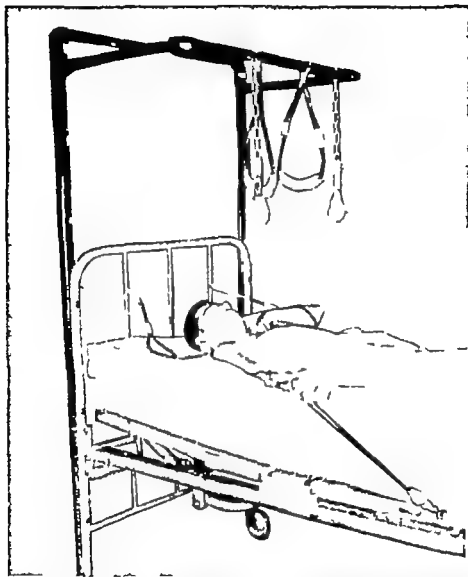


Fig. 10 Loops of rubber tubing for use as self-exercisers by quadriparetics and quadriplegics. Note that the loops not only hang from above but are also attached to the head and side of the bed.

If the patient is a quadriplegic or a quadriparetic a start should now be made in designing and constructing special equipment to facilitate the greatest possible practical use of whatever muscles he has that are still under his voluntary control. A spoon, knife, fork and pencil, for example, may prove usable if made with a large or specially shaped handle. He

can be equipped with and taught to use a rod to operate the keys of a typewriter. In neglected cases spring and turnbuckle splints can be used to stretch contracted finger and elbow tendons and to mobilize these joints if stiff. In particular every effort should be made to develop any remaining triceps finger and wrist flexion and extension that may have been left. In addition exercises to train nonparalyzed pronator muscles as a substitute for a paralyzed triceps so that extension of the forearm on the arm and fixation of the elbow in extension may be attained must be started. Pulley exercises are helpful and in the severe cases loops of rubber tubing of appropriate length hung down from an overhead bar or attached to the head and sides of the bed are very useful as a method of providing a combination of active assisted and passive motion that is entirely within the control of the patient himself (Fig. 10). Exercisers that support the weight of the paralyzed arms during the activity of any weak muscles that still function are especially desirable. A two-branched metal goose-necked frame each branch of which is equipped with weight bearing loops of wide webbing designed to support the weight of the upper arm and forearm respectively may be indispensable. The loops are suspended from cords which run through pulleys and which end in counterbalancing weights. This is an extremely helpful apparatus for facilitating exercise and use of paretic arms. By appropriate modification such an apparatus can be fitted to a wheel chair as well as to the bed. Its use may make such self-care activities as self-reading, turning pages, writing, using a typewriter and the like possible where without its use these activities would either be impossible or would require the services of an attendant that could otherwise be dispensed with.

Later in the convalescence of these patients full use should be made of hoists, electrified wheel chairs, elevators and similar apparatus powered by modern, ultra-efficient electric motors. In this way a maximum of activity is permitted the patient in response to no more effort than is required to push or otherwise move a switch.

In general it may be said that the greater the imagination of the doctor and the patient in inventing substitutes for lost motions in quadriplegia, the greater the reward in terms of increased activity self-care initiative and normal social relations for the victim.

The patient remains in class 2 on an average of from two to eight months.

Class 3 The Stage of Wheel-Chair Mobilization

By the time the patient is ready to enter this stage his bone injury will be healed sufficiently to allow him to sit up either with or without a back splint and to take active exercise. All major diagnostic and reparative surgery will have been completed. His bed sores will be healed and he will have no or at most, only minimal spasm. His bladder will be reflex if the cord injury has been a transection and normal if the cord injury has

band should be attached to a prolongation of the outer upright of the calipers by free swinging, nonlockable hinged joints placed accurately at the level of the trochanter. For patients with paralysis below T1 and above T8 a back brace with an abdominal pad attached in place of and similar to the pelvic band is necessary. The back brace need not come higher than to overlap the lower ribs in back. Its purpose is to act as a substitute for the paralyzed paravertebral muscles and thus permit the chest and pelvis to be moved as a unit. Patients who have cervical injuries and who can sit upright should be provided with a Zimmer-type brace modified to include a pelvic attachment (see page 136). If bilateral calipers can be used on such a patient's legs, and if he is able to stand this brace is used as a separate unit in addition to the calipers with a pelvic attachment. Patients with partial paralysis will have to have their splints designed to meet their individual needs and in accordance with the level of cord or cauda injury.

Wheel chairs must also be provided at this time (see page 247). They should be of light strong metal, collapsible and constructed with the big wheels in the rear and with efficient brakes. Eight-inch front wheels make them easier to handle. The arms should be removable and supports should be provided that can be swung into place behind the calves. Adjustable leg supports will only be necessary for patients with stiff knees or hips. A high back may be essential for quadriplegics. A pad of foam rubber should be used on the seat under the patient's buttocks.

The length of time that the patient spends in this stage will depend solely on his initiative and industry.

Class 4 The Stage of Early Ambulation

The objects to be attained in this stage are an increase in the strength of the patient's crutch muscles, the development of confidence in his ability to keep himself erect and an increasing mobility with the help of his crutches. Until these ends are met it is useless to try to advance further along the road to mobilization. When these ends are attained, he learns the swing-through crutch gait as a preliminary to greater skills if he is paraplegic and as a necessary foundation to the two- and four-point and other simpler gaits if he has only a partial cord or cauda injury.

MAT EXERCISES * These are continued and increased in scope, length and severity over those of the previous class (see also *Class 2 Bed Exercises* page 75).

BRACES AND CRUTCHES * He learns to put on and take off his own braces without help and is fitted to crutches as he has sufficient use of his shoulders, arms and hands.

CRUTCH BALANCING * Crutch balancing exercises—first between parallel bars then against the wall and finally in the center of the room—are learned. Following this he progresses through the shuffle to the swing-to gait, to turns and finally to the swing-through gait and if possible to two- and four-point walking.

RESIDUAL SPASMS. It is during this stage that residual spasms of the trunk and legs may manifest themselves. If they interfere with ambulation they must be dealt with surgically by such procedures as an anterior rhizotomy, tenotomies, tendon lengthening, neurotomies, neurectomies and peripheral denervation by novocaine or alcohol injections. Attempts to correct for the spasm by locking the caliper braces together at the ankles should not be countenanced. This procrastination will not prove successful in the long run and will eventually be replaced by surgical correction of the spasms. At present spasms involving the arms in quadriparetica particularly are virtually impossible to correct other than by persistent exercise when mild Cervical rhizotomy unless of the rootlet variety while it will stop the spasms will also eliminate any accompanying voluntary motion.

PAIN. Complaints of pain should be treated symptomatically only at this stage. Destructive neurosurgical operations are not justifiable at this time.

FACILITIES FOR TRAINING. If facilities for training are better outside the hospital the patient can now be moved to a hotel or boarding house and attend ambulation classes from there. This usually means that he will have to have the help of an attendant to aid him in getting to and from classes. This attendant need not be a trained nurse but rather should be an individual who will act only as a "prime mover" In particular he should not usurp any of the personal-care and mobility skills that the patient has already acquired.

The time spent in this class will usually cover from two to four months.

Class 5 The Stage of Intermediate Ambulation

This stage merges imperceptibly with the preceding stage. It is devoted primarily to increasing the patient's strength and endurance. This is possible only if he has *confidence* the attribute that he should have acquired in the previous class.

AMBULATION. More exercises are increased still further so that he can learn a greater amount of and increasingly complicated ambulation. This permits a widening of the scope of his social and work life. As minimum requirements he learns to turn around, walk sideways and backwards and to mount low curbs and ramps. He learns to get in and out of automobiles. He must be able to wear his braces all day to walk 150 yards without stopping, to ambulate on all kinds of terrain and must walk to and from all meals and other activities. He must be able to cross a mock-up of a standard street within the traffic light's limit, cover 100 yards in a given time, open and close doors, answer the telephone while standing, learn to drive a specially equipped automobile and so forth, in his braces and without evidence of fatigue. Such activities will be at most only partly within reach of the quadriparetic and will usually be impossible for the quadriplegic.

PAIN. If his pain is one that justifies it and that can be expected to respond to that therapy an anterolateral cordotomy at T1-2 segment is

now permissible. Other operations for pain such as lysis of the cauda equina and sympathectomy can also be done at this time if they are indicated. Therapeutic or other use of narcotics should be countenanced.

The time required for this stage will be from two to four months.

Class 6 The Stage of Advanced Ambulation

This is the final stage. It is in this class that the patient acquires *skill*. He cannot do so until he has achieved *confidence*, *strength* and *endurance* the first by the end of class 4 and the other two during class 5. All that has been previously learned in the classroom is now practiced in public, at first with the help of an instructor and later alone. He will gradually dispense with his attendant and increase his initiative and independence to normal. Shopping trips, walks in crowds and traffic, visits to restaurants, actual employment of automobiles, taxis, buses, streetcars and trains, as well as overnight stops in hotels and many other similar activities are all undertaken and perfected. At the very least these must include learning to get up to an erect position from the ground without help, walking over rough terrain, getting up and down all types of stairs with and without hand rails, in and out of all types of chairs and theater seats, moving himself and his wheel chair in and out of automobiles and walking 300 yards a day in addition to other activities. These requirements will, of course have to be modified for the quadriparetics and quadriplegics.

This stage will take from six to eight weeks.

Care of the Genitourinary Tract

Voluntary Control of Urination

This depends primarily on the neural connection between the sacral spinal segments, with their peripheral prolongation, and an organ that is equipped with a wall that can distend but retain within wide limits its capacity to contract, regardless of the amount of that distention. Such functional activity is known as the *stretch reflex* and is an essential characteristic of the urinary bladder.⁸

The bladder receives urine from the ureters in increments of the order of magnitude of drops. When the bladder urine has reached a significant amount, such as 30 cc., its presence stretches the detrusor and provides a sensory stimulus that is transmitted over the parasympathetic nerves to the second, third and fourth sacral spinal segments. This impulse is then routed to an anterior horn cell transmuted into a motor impulse and passed out of the cord (without leaving these segments) by way of the parasympathetic motor nerves. This motor impulse stimulates the detrusor fibers to contract and the so-called internal sphincter to relax, thus permitting urine to escape into that segment of the urethra that is central to the external urethral sphincter. Its presence there acts as a sensory stimulus of a second reflex. This impulse travels toward the spinal cord over the sensory fibers of the

internal pudic nerve (a somatic nerve) where it enters the second, third and fourth sacral segments by way of the somatic sensory roots. It is then routed to an anterior horn cell in these same segments and transmuted into a motor impulse which leaves the cord by way of the anterior somatic roots. Subsequently this motor impulse travels over the motor fibers of the internal pudic nerve (a somatic nerve) and stimulates and thus causes relaxation of the external urethral sphincter. This completes the opening of the passageway from the bladder to the outside, thus ridding the bladder of the urine. However, the contraction of the detrusor muscle that starts the process can be inhibited and the emptying of the bladder postponed up to whatever critical level of fill is peculiar to each individual bladder. Such effective inhibitory impulses can arise from psychic and subconscious as well as conscious levels. The central-nervous-system origin of the first two appears in general to be in the hypothalamic region. The impulses pass down the cord in an unidentified tract and impinge on the detrusor by way of the parasympathetic nerve supply. They are permanently absent in all cases of cord transection, regardless of level, and in all cases of radicular and pelvic injuries that denervate the bladder wall. The sympathetic nervous system as such is not involved. The third, or conscious, source of inhibition is effected through the somatic nerve supply to the external sphincter. Voluntary contraction of the external urethral sphincter and adjoining perineal muscles is caused thereby. These impulses arise in the voluntary motor cortex, travel down the cord in the lateral columns and pass from the cord by way of the internal pudic nerves. These impulses, which act only to close the urethra, do not of themselves inhibit the emptying contraction of the detrusor. They do, however, afford time and possibly act as a stimulant for the subconscious inhibitions of such contractions. Any emptying contraction that is inhibited is followed by elongation of the detrusor muscle fibers, with a consequent enlargement of the capacity of the organ. forcible stretching of the muscular wall of the bladder to a point of maximum elongation of the fibers causes tetanic contractions. These are followed by a paralysis that is mechanical and not neural and that is followed in turn by rupture. Voluntary mediation of detrusor emptying contractions is not possible except through failure to inhibit.

A third source of innervation is found in the wall of the bladder. This is an autonomous nervous plexus such as those of Auerbach and Meissner which are present in the wall of the intestinal tract. Under ordinary circumstances it is assumed that the former has the duty of regularizing and distributing the muscular contractions of the detrusor evenly. With the bladder completely denervated, this plexus assumes control of the detrusor and the internal sphincter. The control is inefficient, however, and will produce only ineffective, short-lived emptying contractions. The sympathetic nervous system has the function of transmitting pain impulses from the bladder. Pathways other than these that transmit any bladder sensations short of pain are not certainly known.

Tidal Drainage Cystometry Bladder Training and Sphincterometry

Tidal drainage cystometry bladder training and sphincterometry are interdependent. When properly used in combination they ensure that every patient who has had an injury to the spinal cord, conus or cauda equina and who is intelligent and co-operative will have infallible twenty four hour control of urination regardless of the severity of the cord injury

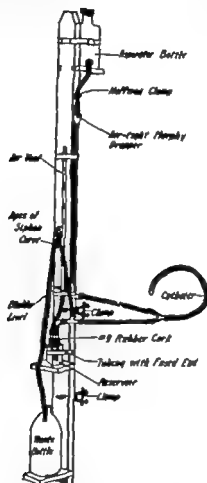


Fig. 11 Original tidal drainage apparatus, now discarded in favor of later models.

TIDAL DRAINAGE Tidal drainage is a method whereby the urinary bladder can be alternately and automatically filled and emptied with the help of irrigating fluid the intravesical pressure constantly maintained at predetermined level puddling of residual urine and overstretching or shrinkage of the bladder wall prevented and the bladder maintained in a close approximation to a physiologically normal state even when completely separated from the suprasegmental part of the central nervous system. The proper use of the apparatus depends primarily on cystometrographic demonstration of the type of bladder to be severed Those hydrodynamic laws that have to do with siphonage determine its activity Its proper functioning depends on

the maintenance of an airtight siphon system during the period of emptying of the bladder

Siphonage is activated whenever a predetermined intravesical pressure has been reached. The siphon is broken and inactivated by the admission of air as soon as the bladder is emptied. The apparatus is thereby left in such a condition that the cycle can begin again. The control of the admission of air into the system was accomplished in the original apparatus by

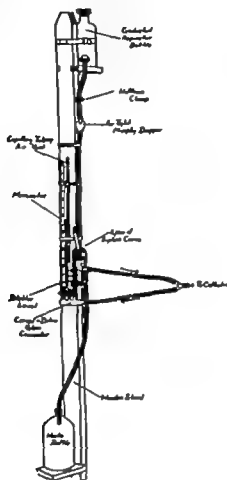


Fig. 1.—A satisfactory tidal drainage apparatus the model that uses the Carrel-Dakin tube.

the use of a water trap. This trap had to be filled before the bladder was filled and before the siphon system could be made airtight. It had to be emptied after the bladder had been emptied before the siphon could be broken. Subsequent improvements led the irrigating fluid directly to the bladder rather than by way of the trap (Fig. 11) and then replaced the trap with a capillary tube. The bore of this tube was too small to permit enough air to enter to break the siphon as long as the system contained any fluid. Through the use of a four-outlet Carrel-Dakin glass connector a built in cystometer was included with this air vent (Fig. 12). Finally

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the air vent and cystometer were amalgamated by the use of a tube with a sufficiently large bore to act as a cystometer the apparatus was given the features of the air vent by being made airtight except for the air that entered and escaped through a hypodermic needle thrust through a rubber connection at its top (Fig. 13) All previous models have been abandoned

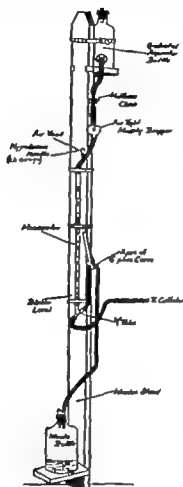


Fig. 13 The best tidal drainage apparatus: the model that uses the hypodermic needle.

in favor of these last two which have been given extensive and thorough trials. Both are entirely and equally satisfactory if properly constructed, set up and used. Neither is adequate unless used in conjunction with cystometrograms made once a week, or oftener if the occasion requires it.

The irrigating fluid should be either 1/20 000 aqueous Zephiran or one-half of one per cent acetic acid solution Potassium permanganate, tap water salt solution and boric acid solution should never be used. The solutions M and G of Suby et al.⁶ should be used only for specific indications.

Construction of the Apparatus As will be seen from Figures 12 and 13

certain parts of the apparatus are common to both models. These are the following

A wooden stand, equipped for attachment to the foot or head of the bed, measuring 6½ feet long by 4 inches wide and with a shelf 6 by 6 by 1 inches at either end and on opposite sides. A centimeter scale marked every 0.5 cm., with its zero at the same level as the patient's pubis, is applied to the side of the upright that the lower shelf is on, the zero indicating the bladder level referred to in the figures and the text below.

A 2300-cc aspirator bottle to which an airtight Murphy dropper that does not have a hole in its side is attached by an appropriate length of rubber tubing, an adjustable compression clamp controlling the rate of flow through the dropper. The aspirator bottle is strapped in place on the top shelf of the stand and the inlet is closed with a sterile gauze or cotton plug.

A waste bottle corked with a doubly perforated stopper containing short pieces of glass tubing in the perforations.

A No. 14 F soft-rubber rectal tube for use as a catheter in male patients, and either that or a No. 14 F soft-rubber mushroom catheter for use in female patients, held in place by appropriate adhesive strapping applied to the penis or the pubis, respectively. It is important that the maximum size of the catheters be limited to No. 14.

To construct the apparatus shown in Figure 12, the following additional material is needed:

A four-outlet (three lateral and one terminal) Carrel-Dakin glass connector.

A piece of glass tubing 70 cm. long with an inside bore of about 3 mm. This is the *cystometer tube* and is attached to the lateral opening that is closest to the closed end of the Carrel-Dakin glass connector by a piece of rubber tubing surrounded by a compression clamp, which is closed when the apparatus is used for tidal drainage and opened when it is used as a cystometer. The Carrel-Dakin connector with the air vent and cystometer tube attached, is strapped to the wooden upright in such a position that the horizontal center line of the connector is 5 cm. below the bladder level and so that the cystometer tube lies beside the centimeter scale on the wooden upright.

A Y tube. The aspirator bottle and attached airtight Murphy dropper are connected by a sufficiently long piece of rubber tubing to one arm of the Y tube, whose upright is inserted in the open end of the catheter. Another equally long piece of rubber tubing is led from the other arm of the Y to the open end of the Carrel-Dakin connector.

A length of small (inside diameter 3 to 5 mm.) rubber tubing, attached to the third side opening of the Carrel-Dakin connector. This tubing is carried upward to some point higher than the bladder level, passed through a loop of adhesive (this makes the *siphon curve*) and then led downward and attached to one of the glass tubes in the cork in the waste bottle. The other glass tube remains open. The waste bottle stands on the lower shelf of the wooden upright.

To construct the apparatus shown in Figure 13 the following material is needed in addition to the basic equipment listed above:

A piece of glass tubing 70 cm. long, with a bore of about 3 mm. (the *cystometer tube*) whose upper end is attached by a short piece of rubber tubing to the outlet of the airtight Murphy dropper.

A hypodermic needle with a patent bore, which is thrust through one wall of the rubber tube. This is the *air vent*.

A Y tube, one of whose arms is joined by rubber tubing to the lower end of the cystometer tube the upright being connected to the open end of the catheter by means of rubber tubing and glass connectors. The cystometer tube and the attached Y tube are then strapped to the wooden stand in such a way as to place the former beside the centimeter scale with its lower end 5 cm. below the bladder level.

A length of small rubber tubing (inside diameter 3 to 5 mm.) which is led upward from the other arm of the Y tube to some point higher than the bladder level, passed through a loop of adhesive (this makes the *siphon curve*) and then led downward to be attached to one of the glass tubes in the cork in the waste bottle, the other glass

demonstrated that this is slow enough to reproduce the physiologic response to the normal filling that takes place by way of the ureters. In every case a catheter that is not of the Foley type is fastened in place in the bladder. The aspirator bottle in either type of tidal-drainage apparatus is replaced by a cylindrical glass container graduated in 25 cc. amounts. The siphon curve is raised to 50 cm. above the bladder level. The cystometer is first filled with an appropriate bland fluid and all air bubbles evacuated, after which it is closed off at the point of attachment to the catheter by a clamp. The bladder is then emptied, the catheter is attached to the cystometer and the clamp is loosened. The compression clamp between the cylindrical container and the airtight Murphy dropper having been opened so that fluid flows at the proper rate of 90 drops a minute and the meniscus in the cystometer tube having been adjusted to the bladder level by the addition of any necessary fluid, it is possible to fill the bladder and read the intravesical pressure simultaneously. The pressure is read in connection with the height above zero of the meniscus in the cystometer tube. The amount of fluid that is in the bladder at any one time is determined by reading the fluid level in the glass cylinder. Changes in this level of as little as 10 cc. can be approximated with sufficient accuracy to justify recording. Each change in intravesical pressure—whether up or down—that is noted on the cystometer is recorded, and opposite it is placed the amount of fluid that has run over into the bladder up to that point. An additional notation is made whenever there is leakage about the catheter or whenever the cystometer tube or siphon curve overflows. Recordings are continued until 400 cc. of fluid has run through the apparatus, unless there is reason to fear reflux up the ureters, in which case smaller amounts and lower pressures are used. The catheter is then disconnected from the apparatus, and the bladder is allowed to empty itself with the open end of the catheter held at zero level. This urine is collected and measured. When this process is completed the catheter is lowered and any residual urine that flows out by siphonage is also collected and measured. The operator will then have two parallel columns of figures (one being intravesical pressure readings and the other intravesical cubic centimeters of fluid) as well as the amounts of the residual urine and the urine in the bladder at the close of the procedure.

Interpretation. From these data necessary information concerning bladder function can be obtained. The presence of *emptying contractions* is indicated when the intravesical pressure reaches or exceeds 50 cm. of water when there is leakage around the catheter or when either the cystometer or the siphon curve overflows. By noting the amount of fluid that has been delivered to the bladder between succeeding emptying contractions, it is possible to determine the periodicity and frequency with which the contractions occur.

The *curve of basic tonus* can also be observed. By subtracting the lowest intravesical pressure from the highest and noting the amount of fluid that has gone through the apparatus a fairly accurate estimate of the shape of

the curve of basic tonus can be obtained. This is a function of the irritability of the detrusor muscle.

The *minimal capacity* of the bladder is the amount of fill that has collected between the first and second emptying contractions.

The *residual urine* is best expressed in percentage of fill. To obtain this the amount of urine collected by siphonage with the catheter opening held below the zero level (residual urine) is divided by the sum of the amount of fill (usually 400 cc.) and the amount of urine collected from the bladder at zero level at the close of the procedure.

Temporary elevations of intravesical pressure that neither rise to the height of an emptying contraction nor cause leakage about the catheter are *aborted emptying contractions*.

The presence of an *anal or bulbocavernosus reflex* is determined last of all. The test is made by pricking either the perianal region or the glans penis with a pin and observing whether or not there is a reflex contraction of the anus. If there is a contraction the reflex is normal. If there is none it is abnormal.

By plotting the two columns of figures (centimeters of intravesical pressure and cubic centimeters of intravesical fluid) on co-ordinate paper a graph of the bladder activity during this experimental fill can be drawn. This is not necessary for adequate interpretation, however. Figure 14 depicts the composite result of cystometric data collected as described above from observations on the bladders of normal persons.

In the interpretation of cystometrograms certain information is essential; other data are not essential but may possibly be useful, and some are misleading in an estimation of the degree of recovery of bladder function. The *essential cystometric information* is as follows: the presence of emptying contractions—only normal or reflex bladders develop the classic emptying contractions; the curve of basic tonus varying from the flat curve of atonicity to the steep gradient of hypertonicity—the former may be present in normal bladders and will be present between the emptying contractions in reflex bladders; and the latter occurs as described below: an anal or bulbocavernosus reflex, which is present only in the absence of spinal shock and when the internal pudic nerve and its spinal-cord connections are intact, indicating either normal voluntary or abnormal involuntary reflex control of the external urethral sphincter; and the capacity of the bladder which will be a minimal figure varying from the large amount that is limited only by the elasticity of the bladder wall and is collected in atonic bladders to the small amount that is present in bladders that are hypertonic, hypertrophied and shrunken or infected—a minimal capacity below 150 cc. is abnormal.

It is useful to know whether or not aborted emptying contractions are present, how much residual urine (expressed in percentage of fill) is present, and if the patient can tell whether his bladder is empty or full and needs to be emptied.

It may be misleading to determine at what point the desire to void occurs

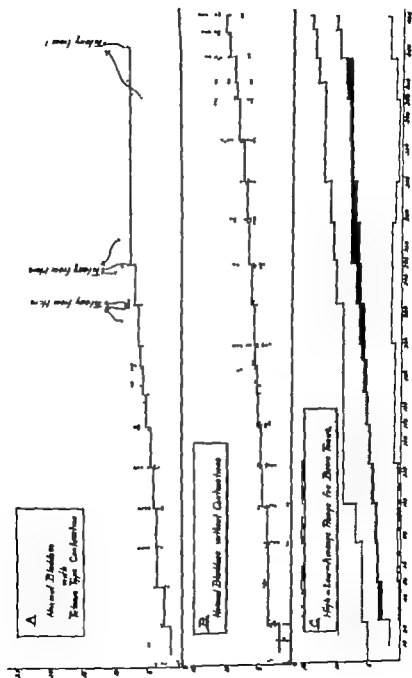


Fig. 14. Graphs of normal bladder activity as measured by cystometry

and at what point pain develops. The first is a function of the patient's ability to inhibit emptying contractions and is motivated by such a wide variety of stimuli—varying from inability to urinate while in bed or in the presence of another person to the effect of a total cord transection—that conclusions based on it not only are unreliable but may lead one astray. The sensation of pain in the bladder wall, if present, means that the organ is being dangerously overstretched. If not present, it may mean the same thing only this time that the danger will be greater because it is unrecognizable. In addition, it may be pointed out that any interpretations made by the patient, and that therefore require an intact nervous system, are valueless in the face of an anatomically or physiologically transected spinal cord. The

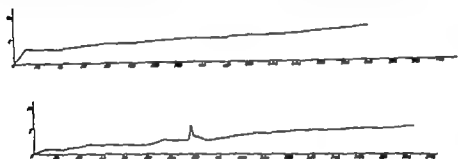


Fig. 15 Graphs of the activity of an atonic bladder as measured by cystometry. The upper graph was made 10 hours and the lower graph 43 days after a cord injury. The latter however demonstrates a redevelopment of the atonicity following an acute infection and the recurrence of spinal shock.

observer who is accustomed to rely on such observations in his clinical work will find himself without them and at a loss in the type of case in which he most needs cystometrography.

When a human spinal cord is transected either anatomically or physiologically below the fourth cervical and above the second sacral segment, in addition to a complete anesthesia and a total loss of all voluntary motor control below the level of cord injury, the immediate response of the bladder is to become atonic (Fig. 15). The detrusor muscle does not contract at all. The internal sphincter closes tightly and remains closed and unresponsive to any physiologic stimuli during this phase. The external sphincter is relaxed and the anal and bulbocavernosus reflexes are abolished. Any emptying that occurs is only the result of leakage through the contracted internal sphincter and takes place because of the elasticity of the bladder wall and not by virtue of any contractile effort on its part. The bladder capacity is limited only by this elasticity. Bladders that have advanced beyond this stage toward recovery will regress to it in the presence of spinal shock, exhaustion, sepsis, toxemia and hypoproteinemia (Fig. 15). The length of time that a bladder remains in this atonic stage depends on these complications, the severity of the injury and the efficiency of the treatment.

The next stage toward recovery is characterized by the development of

ineffective aborted emptying contractions (Fig. 16). The level of basic tonus is slightly higher than that in an atonic bladder. The internal sphincter is tightly closed and opens only partially in response to the inefficient, aborted emptying contractions of the detrusor muscle. The bulbocavernosus and anal reflexes may return. There are no emptying contractions. This stage may be extremely short and will be missed if cystometrograms are not made frequently enough. These bladders discharge small amounts of urine frequently. Their capacity is at first large but becomes increasingly smaller as hypertrophy of the muscular wall develops. These bladders regress to the atonic stage for the reasons given above. The length of time that the bladder remains in this stage again depends on any complications and the efficiency of treatment but not on the severity of the injury. This is the autonomous stage so called because it is believed, but not proved, that the neural control is exerted by way of an intramural autonomous plexus.

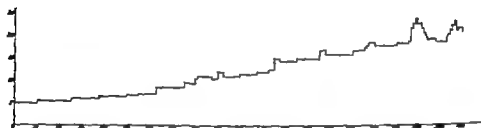


Fig. 16. Graph of the activity of an autonomous bladder as measured by cystometry 16 days after a cord injury. Note the aborted emptying contractions.

The third stage toward recovery is the hypertonic. This is characterized by something approximating tetany of the detrusor muscle (Fig. 17). It is in a nearly constant state of contraction; the curve of basic tonus is steep and the intervals between emptying contractions are extremely short. The internal sphincter reacts physiologically, opening with each emptying contraction and closing in the intervals. The external sphincter responds reflexly and the anal and bulbocavernosus reflexes are present. The bladder capacity, as measured by the amount of urine stored between emptying contractions, is small. This stage is often of short duration and either regresses toward atonicity or progresses to the reflex stage. An acutely infected but otherwise normal bladder may give a cystometrogram that is indistinguishable from one made during this stage. Normal uninfected bladders exhibit these characteristics just before emptying if they are distended beyond normal limits but have not been so stretched as to have become mechanically incapable of contracting (Fig. 14).

The final stage is that of the reflex bladder (Fig. 18). In small children who have not yet learned to obey the dictates of civilization this represents normal bladder activity. Increase in capacity and training in the inhibition of emptying contractions produce the control that is apparent later as normal adult micturition. Under certain conditions, however, this inhibitory ability can be lost again, the normal bladder reverting to reflex activity in

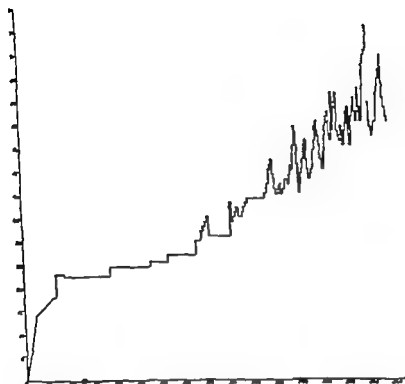


Fig. 17 Graph of the activity of a hypertonic bladder as measured by cystometry 5 days after a cord injury. Note the high basic tone and the tetanic type of emptying contractions.

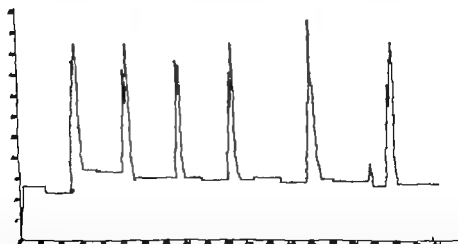


Fig. 18 Graph of the activity of a reflex bladder as measured by cystometry 7 months after injury. Note the regularity of the emptying contractions, the relatively low basic tone and the complete emptying of the bladder at each contraction. This bladder is the same as that from which the graph in Figure 16 was made.

the absence of any physical impairment of its nervous connections. Emotional stress such as extreme fear of death or bodily harm may so relax the inhibitory effect exerted by the higher centers that the person experiencing such an emotion finds himself—if the bladder has not been emptied just previously—wet with urine once the danger is passed. Coma if sufficiently deep produces the same chain of events if it is less profound, and even though the victim is out of conscious touch with his surroundings, inhibitory control continues to be exerted however and the bladder is prevented from emptying (see p 103). Convulsive seizures with the profound coma and loss of "sphincter control" of grand mal, on the one hand, and the momentary unconsciousness without soiling of the petit mal attacks, on the other as well as the variations between, illustrate these phenomena in all degrees.

Reflex bladder activity is also a function of the anatomically and physiologically transected cord. Under such conditions it is the best end result that can be attained, and bladder training as described below is merely a method of making practical use of this phenomenon. It is this activity that has been referred to in the past as the "automatic bladder." In nontransecting injuries, it is not and should not be considered an end result. In these injuries, the activity is analogous to that of the reflex bladder of the normal infant. Further training enables the patient to regain the normal inhibitory control just as the child does as he grows older. In this reflex stage the detrusor muscle contracts reflexly and completely in response to the sensory stimulus provided by the stretch of a critically filled bladder. During the fill and before the accumulation of this critical amount of urine any emptying contractions are aborted in favor of a stretch reflex and consequent increase in the capacity of the organ. The internal sphincter remains closed until the emptying contraction takes place when it opens and permits urine to escape into the posterior urethra. This in turn, acts as a sensory stimulus that causes the external sphincter to relax reflexly from its previously contracted closed position. The bulbocavernosus and anal reflexes are present. If the cord injury is nontransecting, the patient also has the ability to relax and contract the external sphincter voluntarily. If the injury is a transecting one this voluntary control is permanently lost. If the bladder has not been allowed to shrink during the early stages of its recovery from atonicity its critical capacity varies from 200 to 400 cc or more. When it does empty all the contents are expelled. Reflex emptying in cases with a transected cord may also be initiated—regardless of the amount of the contents—by contraction of the abdominal muscles, stimulation of the abdomen, thigh and other areas of the leg, catheterization, movement of the bowels and change in position. Facilitation of micturition as a part of the "mass reflex" is no more than an indirect and coincidental effect. Regression toward atonicity takes place in the presence of infection, toxemia, exhaustion, a major operative procedure, hypoproteinemias and so forth. A decrease in capacity—and hence an increase in the number of emptying contractions—develops in the presence of cystitis, vesical calculi and prolonged suprapubic drainage. Urinary retention despite the presence of

otherwise efficient emptying contractions has been shown to be caused by hypertrophy or fibrosis with a pseudo-median bar formation of the internal sphincter and by unpredictable spasm of the external sphincter. It is probable that this retention has its origin, at least in part, in injudicious early treatment of the bladder and is a complication of too prolonged and constant or suprapubic drainage.

Destructive lesions that are complete and involve the sacral segments or roots of the spinal cord or the parasympathetic plexuses, and hence completely denervate the bladder with regard to its central connections, force the bladder to depend on its intramural plexus for neuromuscular control. This ability develops after the bladder has recovered from the atonic stage. The resultant bladder activity duplicates that described above under the term "autonomous bladder" (Fig. 16). This is the best physiologic end result that can be obtained in such cases. Because of the total denervation it is impossible to impose bladder training on these patients. That does not mean, however, that the patients must accept a contracted bladder that will hold only 30 or 50 cc. and that leaks almost constantly. Even in the face of total denervation, the bladder capacity can be maintained at normal by the prompt use of tidal drainage in the preconvalescent bed stage. When the patient is ambulant, unailing twenty-four hour control can be established if the patient continues to wear an indwelling catheter and very often something that approximates reflex emptying in an inefficient way can be developed to such a degree that the catheter can be dispensed with. During the day the catheter is closed by a clamp except when the bladder is emptied at intervals of three or four hours. During the night, while the patient is in bed, the catheter is opened and attached to a tidal-drainage apparatus with the siphon curve set at 10 to 12 cm. The bladder wall is thus kept from hypertrophy and contraction, and the patient is enabled to keep himself dry and clean at all times. The catheter is removed, cleaned, resterilized and replaced aseptically once a week and the apparatus taken down, cleaned, resterilized and reassembled after the same interval. One half per cent acetic acid or 1:20,000 aqueous Zephiran solution is used as an irrigating fluid. Any person of ordinary intelligence can without difficulty be taught the proper care of himself and this apparatus.

Impulses reaching the bladder by way of the sympathetic (as distinguished from the parasympathetic) and somatic nervous systems have no influence on the function of storage or discharge. Denny Brown and Robertson's original contention that the effect of the sympathetic nervous system on the bladder concerns the transmission of pain from that organ appears to be confirmed.

BLADDER TRAINING Bladder training should be necessary only in patients with anatomically or physiologically transected cords. It will be effective only if the patient takes an intelligent interest in the problem and co-operates. If the bladder has been properly cared for from the time of injury such training is not necessary with nontransecting cord injuries. It is ineffective in patients with destructive lesions of the sacral cord or roots and of the

parasympathetic plexuses. It cannot be imposed on and is not successful in, any patient who is in spinal shock, who has an uncontrolled "mass reflex" type of spasm who is mentally deficient or unco-operative or who has a suprapubic or perineal cystostomy bladder obstruction from any cause an overstretched bladder with a wall that is mechanically incapable of contraction because of constant distention a bladder that has a wall that over reacts to minimal stretch stimuli, an infected bladder vesical or renal calculi, pyelitis or pyelonephrosis general sepsis, toxemia exhaustion debilitation, hypoproteinememia avitaminosis, large unhealed bed sores or the like. The bladder activity must be of the reflex type and the bladder capacity at least 200 cc. In patients with an active mass reflex the lower half of the abdominal wall must have been paralyzed either because of appropriate thoracic root destruction as part of the injury or following operative bilateral anterior dorsolumbar rhizotomy. To prevent genitourinary infection the fluid intake must not be allowed to fall below 3600 cc. in twenty four hours during the period of training. Bladder training is much easier when the patient is ambulant in splints and with the aid of crutches, but its initiation should not be postponed until this stage is reached. The patient must have sufficient use of his hands to clamp and unclamp the catheter must have a watch must understand the elements of the problem he is faced with must be co-operative and must be willing to assume full responsibility for the solution of that problem. With these requirements met, all that is necessary is patience and perseverance on the part of both the patient and the doctor.

Control of micturition is possible in the presence of a transected cord because the bladder if not otherwise altered, can be distended to a point where it contains from 200 to 400 cc. of urine before emptying and because an emptying contraction can be initiated as a reflex response to appropriate sensory stimuli at any time previous to that point. By appropriate regulation of the fluid intake the time that elapses before this amount is collected in the bladder can also be predetermined. This will vary individually and from hour to hour during the day and night in accordance with the efficiency of the early treatment and with the filtration characteristics of the patient's kidneys. By reflex emptying of the bladder under controlled conditions immediately before the contents reach the critical level, soiling with urine at unexpected times can be prevented.

The first stage in the training is to condition the bladder so that it will distend to the point where it holds 200 cc. or more without developing an emptying contraction. This phase can and should be started while the patient is still in bed and as soon as the bladder activity has become reflex in type. To do this the catheter is left in place but disconnected from the tidal-drainage apparatus and clamped off for an hour and a half at a time. At the end of each of these periods the catheter is opened, the open end placed so as to empty into a urinal and the bladder made to contract by appropriate reflex stimulation such as massage of the abdomen scratching of the thigh or any similar procedure that is effective. Forceful expulsion of

the contents by pressure through the abdominal wall should not be practiced under any circumstances. The fluid intake is adjusted so that no fluid is taken between 7 00 P.M. and 7 00 A.M. During the other twelve hours 300 cc. should be ingested every hour preferably on the hour. This fluid intake remains the same throughout the training period and thereafter until full adjustment and control are obtained. The patient should be held responsible for draining his own bladder at proper intervals during the day. At night the catheter should be attached to the tidal-drainage apparatus. After he is able to go for a week without leakage between emptyings, the interval should be increased to two hours. The process is repeated until the same requirement can be successfully met. After this, the interval is increased to two and a half hours and then to three hours. The greatest difficulty is usually encountered in making the change from an hour and one half to two hours. If leakage occurs at that time or at any other change the patient should be put back on the next shortest interval for another week or so and the shift upward tried again.

When the patient can successfully manage a three-hour interval without leakage he should be taken off tidal drainage at night. Instead, he should be awakened and made to empty the bladder every two hours until a week has passed without leakage, and subsequently every three hours. He will then be able to retain the bladder contents for three hours at a time without leakage, day and night, but the catheter will still be in place. The activity of the detrusor muscle has been conditioned, but not that of the sphincters. To attain that end, the catheter should be removed entirely and the time interval between emptyings cut back again to an hour and a half. The intervals are then increased as indicated above but without the catheter. The bladder should be catheterized once every twenty four hours, immediately after it has emptied itself, and the amount of residual urine noted. If the residual urine is 30 cc. or less or if it steadily decreases in amount each day the training may be continued. If, however, the residual urine increases or remains the same, the training should be interrupted to make sure of the cause. This will require sphincterometric studies and a cystometrogram. It is during this stage that obstruction caused by a spastic external or hypertrophied internal sphincter becomes evident if it exists. Such a condition occasionally requires injection of the pudic nerves or resection of the internal sphincter but this should not be done until a serious effort has been made and enough time allowed to elapse for the sphincters to correct their own spasticity. Any other obstruction to emptying must be corrected at once and before the training can be continued. It is at about this time also that the patients begin to be ambulant. This change from the recumbent or sitting to the erect position also alters the bladder activity. It is often found that control of micturition which is entirely satisfactory with the patient erect fails miserably when he returns to bed. Increased exercise such as greater ability in using crutches, may also cause temporary intermittent leakage. Persistence in practice and repetition of the earlier training stages, even perhaps to the extent of catheter replacement, will overcome all these diffi-

cultures sooner or later particularly if an increase in ambulation is insisted on. It is also helpful if the patient makes a practice of using the toilet rather than the urinal at each bladder emptying. The position of the body and possibly also the psychology of urinating in such surroundings appear to be conducive to greater efficiency in micturition. In especially difficult cases much help can often be obtained by making the patient keep his own intake and output chart. This should include a time schedule as well.

Complications Once the patient has reached the stage where he can go for three hours without urinating or leakage during both day and night, he can be allowed to go through the night without being awakened at all or at most, only once. The daytime interval can also be extended from half an hour to an hour if the need arises. Under no circumstances should the interval between drainages be shortened such a procedure will quickly cause regression of bladder function. Any unexpected break in efficiency should lead to prompt investigation of the cause. For example a vesical calculus that had been entirely asymptomatic until the patient was well past the first stages of ambulation is a common cause of such regression. Sudden inability to control micturition has been found to have been caused by a small papilloma that had developed and was missed in spite of previous cystoscopic examinations while the patient was under treatment for a cord injury. Removal of the stone and fulguration of the papilloma permitted both patients to regain twenty-four-hour control of micturition.

Successful bladder training can be acquired by any patient with a transected cord if the requirements discussed above are met. It is acquired much more easily and rapidly however if the bladder is kept in a state of physiologic normalcy during the immediate post-injury convalescence by the proper use of tidal drainage supplemented by the necessary number of cystometrograms. As pointed out above, an automatic bladder although better than a contracted or permanently drained bladder cannot be considered a satisfactory end result. It is merely a preliminary stage in the return of bladder function and should be recognized as a condition that can be transmuted into unfailing twenty four hour control of micturition if the physician is alert to the possibilities and the patient intelligent and co-operative.

Patients with denervated bladders (due to destructive lesions of the sacral cord or roots or parasympathetic plexuses) have the same control but must be willing to wear a catheter constantly and to use tidal drainage at night. They are trained in the manner described above to increase the intervals between bladder drainages from an hour and a half to three hours, except that they never reach the stage of training without catheters or the stage in which they are not awakened at night. It is possible that resection of the internal sphincter will eventually prove to be the solution that will permit removal of the indwelling catheters and abandonment of the tidal-drainage apparatus in these cases.

No satisfactory method of treating the urinary difficulties of patients with paralyzed hands has been evolved, other than to have an attendant

open and close the catheters during the first part of the training period. If that difficulty can be overcome there is no reason why such patients should not eventually attain bladder control once the catheters can be removed. After that the reflex emptying that follows sensory stimuli set up by scratching the abdomen or thigh will be within the scope of their limited activities. Patients with nontransected cords with hand paralysis traceable to cord changes associated with hematomyelia will have normal bladder activity if their bladders are properly treated from the start. The only problem is to obtain urinals that they can use when necessary. With normal control of micturition accidents can usually be avoided because of the leeway permitted by virtue of the ability to inhibit an emptying contraction for a reasonable and possibly a considerable period.

Patients with cauda-equina injuries that do not denervate the bladder may nevertheless have sufficiently disabling bladder symptoms to prevent a normal social and work life. Cystometrically the only variation from normalcy is a decreased capacity and evidence of hypertonicity in that emptying contractions develop more frequently than normal and the curve of basic tonus is steeper than normal. The bladder symptoms are usually extreme urgency, frequency, dribbling and unexpected major leakage. Varied other symptoms of radiculitis also develop, the usual ones being pain alone or in combination with the type of urinary difficulty described above. Clinical evidence suggests that to correct the bladder symptoms the radiculitis must be relieved and the bladder stretched if it has shrunk. Pre-operative investigation of the dynamics of the spinal cerebrospinal fluid is essential and usually reveals a partial and often complete dynamic block. If it can be done at all, relief of the radiculitis is accomplished by a decompressive laminectomy in which the five laminae overlying the cauda equina are removed, the dura is opened for the full length of the wound and any neurolysis that may be possible is carried out. The wound is then closed without drainage and without closing the dura. The patient is put on tidal drainage adjusted to meet the requirements of the cystometric findings and to stretch the bladder if necessary. This is continued until the capacity has been raised to over 200 cc. after which bladder training as described above is instituted. If the detrusor hyperactivity is not relieved by such means then recourse must be had to injection of the sacral nerves as described on page 104.

Fluid Adjustment. In certain instances and especially in patients the care of whose genitourinary tract has been neglected in the early months after their central-nervous-system injury, a satisfactory fluid adjustment proves to be virtually impossible on account of the irregularity of urinary secretion. Despite the fact that all fluid is ingested in 300-cc. amounts every hour on the hour for the twelve waking ones, the bladder when emptied, will sometimes contain as little as only 150 cc. and sometimes as much as 700 cc. In the latter instance and in similar instances in which the contained urine is even less in amount, its capacity will have been exceeded or its walls badly overstretched. This either produces leaks with an increased

incidence of bed sores, or damage to the bladder wall and, in either case, may effectively prevent bladder training. Figure 19 demonstrates this irregularity graphically. The solution of this problem is simple and obvious. If the patient does not have a chronic pyelitis and if the reduction of his fluid intake will not light up a dormant one the total fluid intake can and should be cut from 3600 cc. to about 2400 cc. for the twelve waking hours.

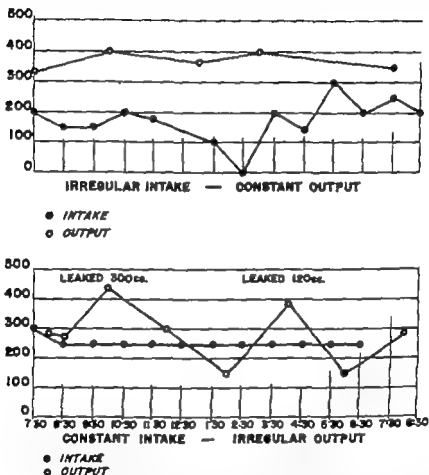


Fig. 19 Fluid intake and urinary output graphs. Note the regularity of output that has been attained by making the intake irregular

The patient should then chart his urinary output against his fluid intake and, figuring two hours as the average time necessary to get the fluid taken by mouth through the kidneys and into the bladder cut down his fluid intake two hours before each peak of fluid output and add those amounts to the fluids ingested two hours prior to the low points of his fluid output. This as shown in Figure 19 levels off the urinary secretion, tends to do away with overfilling and underfilling of the bladder eliminates leakage and thus permits bladder training. If the patient is given the responsibility for making this adjustment he will usually accomplish it in two or three days. Having learned by practical experience how to deal with this phase of his

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daily life he can be counted on to continue to do so thereafter and will thus assure his own welfare to a degree that the doctor would be unable to accomplish unaided

THE USE OF TIDAL DRAINAGE AT HOME. At times it is advisable to provide for the use of tidal drainage in the patient's home. This is particularly true when the patient has sustained such an injury as to denervate his bladder, create an autonomous bladder and require the constant use of an indwelling urethral catheter. Such patients can care for their bladder-drainage needs during the day while active and out of bed by regular emptying of their bladder at three hour intervals. Their bladders will be kept in better condition, run less risk of infection, cause less of a laundry problem and permit sounder sleep if the catheter is attached to a tidal-drainage set at night, however. Anyone of ordinary intelligence can be quickly taught to take down, sterilize and reassemble the apparatus, which can be attached to either the head or the foot of the bed. Weekly supervision by a visiting nurse and monthly checking of the apparatus and of a specimen of urine by a doctor assure its efficient functioning. This can be verified by an intravenous pyelogram twice a year. Cystometrograms can also be done as indicated. The best irrigating solution to use under such circumstances is 1:20,000 aqueous solution of Zephiran.

THE USE OF TIDAL DRAINAGE IN ASSOCIATION WITH CRANIOCEREBRAL INJURIES. Care of the genito-urinary tract in the presence of a craniocerebral injury is more a matter of recognizing the need for such care than the solution of any problem in respect to the methods to be used. A study of the mechanism of normal micturition will demonstrate that it is essentially a spinal-reflex mechanism that can be brought under control by certain higher centers in regard to inhibition of function. The application of this knowledge to the varied loss of cerebral function that accompanies craniocerebral injuries is the key to the care of the genito-urinary tract when such an injury is present. As long as the patient retains sufficient contact with his environment, even in the presence of apparent unconsciousness and failure to respond to noxious stimuli, to realize that he is not in a situation suitable for urination for so long will he inhibit the bladder-emptying contractions and refuse to urinate. Thus the bladder becomes more and more distended, until finally the sphincters are forced in spite of the attempted inhibition and enough urine escapes to relieve the intravesical pressure but no more. As the bladder fills again this same process is repeated. Because of the pain, discomfort and frustration the patient is restless and irritable, attempts to get out of bed and is difficult to handle. He is suffering from *overflow incontinence* and his symptoms will be largely relieved by catheterization. Any such comatose patient who passes small amounts of urine frequently must have his bladder emptied forthwith. If the patient's coma lessens from this level, he again becomes able to indicate his need for the urinal and is again capable of something approaching normal bladder control.

If the patient's coma deepens to the point where he no longer has any contact whatsoever with his environment, his ability to inhibit emptying

contractions is lost. The bladder reverts to a pure spinal-reflex mechanism. This is the type of bladder function that is present in small untrained children and in patients who have reached the proper end stage (as far as their bladder is concerned) of a transection of the spinal cord above the level of the sacral segments. In these patients the bladder does not empty until it has been distended and its wall has been stretched to the point of fill that is critical for that particular bladder. When this point is reached an emptying contraction is completed and the bladder is *entirely* emptied. These patients pass large amounts of urine infrequently and are quiet and deeply comatose.

Observation of the type and amount of bladder emptying in unresponsive patients with a craniocerebral injury will yield valuable prognostic evidence. A change in bladder emptying from overflow incontinence to reflex emptying will indicate an increase in the depth of unconsciousness and will point toward an increasingly bad prognosis. The reverse, of course, is of good prognostic import.

Patients who are unable to indicate their need for a urinal do better if they are placed on tidal drainage at once. The siphon curve should be set as for a normal bladder. The apparatus should be continued in use until the patient is again able to communicate his needs in a normal manner. The irrigating fluid should be 1:20,000 aqueous Zephiran solution.

THE HYPERACTIVE DETRUSOR. This condition is usually first recognized when attempts are made to install effective bladder training, either with or without a catheter. The commonest cause of such uncontrollable hyperactivity is, or was, the presence of a so-called "mass reflex." The bladder activity is a secondary effect of this in that it results from the continued irritating stimulation applied to it every time the abdominal muscles contract. This not only renders the bladder more sensitive to sensory stimuli but actually sets up a conditioned reflex through which the bladder is trained to empty itself in response to a minimal rather than a maximal fill. This condition is easily demonstrated by cystometrogram and cannot be corrected until after the mass reflex activity of the back, abdominal and leg muscles has been destroyed. If the bladder has been kept stretched and free of infection by the proper use of tidal drainage, the completion of such a corrective procedure will be all that is required to permit proper bladder training. However, if the bladder has been or is infected, if there are stones present or if the "mass reflex" has been allowed to continue uncorrected for six, eight or twelve months or until an abnormal conditioned emptying reflex has become established, it will be impossible to correct the detrusor overactivity and oversensitivity without resorting to other more direct measures.

The most effective of these appears to be a combination of stretch of the bladder wall by tidal drainage and repeated temporary denervation of part of the detrusor. This appears to destroy the abnormal new conditioned emptying reflex that has been set up and allows the filling and emptying of the bladder to revert to its original rhythm.

Temporary denervation of the detrusor is accomplished by the injection of 5 to 10 cc. of 1 per cent procaine solution into certain of the foramina of exit of the sacral roots. The number and identity of the roots chosen to be injected depend on the amount of denervation desired and on the all important fact that the solution must not be injected into both third sacral roots simultaneously. It is usual to inject the second and fourth sacral roots on one side and the third sacral root on the other side reversing the distribution at each subsequent injection. From two to six or ten injections may be necessary to obtain the desired result. They should be repeated every two to seven days and should always be preceded and followed by a cystometrogram. The anal reflex may be diminished or abolished temporarily by this procedure.

The technic is as follows: The patient is placed flat on his abdomen. If the patient is anesthetic in the lumbosacral area because of his injury no local anesthesia will be necessary. If he is not, it is better to carry out the procedure under Pentothal sodium, although it can be done under local anesthesia if necessary. The fourth-fifth lumbar, the lumbosacral and the first-second sacral interspinous spaces are identified by inserting a needle in each. This gives the operator a constant check on the position and direction of the vertical midsacral line. The posterior inferior spine of the ilium is identified on one side and a needle is inserted one finger breadth medial to this point, pointing slightly upward and slightly inward. The point should be felt to enter easily the first sacral foramen. The procedure is repeated on the other side. If the dura is perforated and cerebrospinal fluid escapes, the needle should be withdrawn enough to stop the flow. Roughly a finger's breadth beneath the point of entry of the first needle and somewhat closer to the midvertical line will be found the second sacral foramen, similarly below that and still closer to the midvertical line will be found the third and then the fourth. The needles will have to be pointed more and more upward and somewhat more medially to enter each succeeding intervertebral foramen. When three needles are properly in place on each side from 5 to 10 cc. of a 1 per cent solution of procaine should be injected into the second and fourth foramen on one side and in the third on the other after which the needles can be withdrawn. Before each injection the needle should be aspirated for cerebrospinal fluid or blood; if either can be obtained its position should be changed before injecting the anesthetic. If the injection is successful, a cystometrogram done after the injection should demonstrate an increased bladder capacity, a flatter curve of basic tonus, better and more nearly normal spacing of emptying contractions, no residual urine and a diminished or absent anal or bulbocavernosus reflex.

Sphincteric Complications

THE HYPERTONIC OR SPASTIC EXTERNAL URETHRAL SPHINCTER. The same conditions that frequently lead to hyperactivity of the detrusor may lead also to hypertonicity or spasticity of the external urethral sphincter. This usually occurs alone but may be found as a concomitant of a hyper

tonic detrusor or in conjunction with hypertrophy of the internal sphincter

It should be emphasized at once however that *spasm* of the *external* urethral sphincter has no necessary connection with *hypertrophy* of the *internal* urethral sphincter. In the present state of our knowledge the spasm of the external sphincter is simply an example of muscular over reaction to a minimal sensory stimulus. There is no element of hypertrophy in it. It characteristically appears in partial cord injuries with or without a "mass reflex." It appears to be the same type of conditioned reflex that is

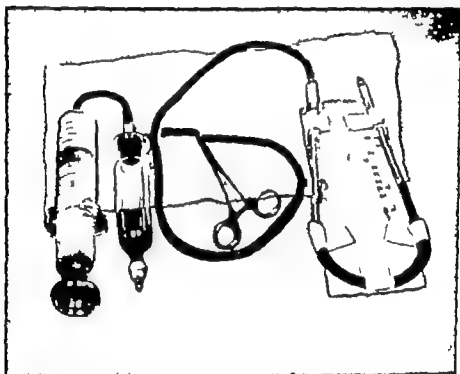


Fig. 20 Sphincterometry apparatus. From left to right, 50-cc. glass syringe perforated rubber cork in the neck of an Asepto syringe, the tip of which passes through the rubber attachment designed for use in the female urethra, a glass T-tube which attaches the manometer connection to both the 50-cc. syringe and the Asepto syringe; clamp on thick-walled, small-bore rubber tubing which connects the glass T-tube with the mercury manometer mercury U-tube type manometer

exemplified by the hyperactive detrusor and it develops in a similar way and from similar causes. It is demonstrable in the majority of instances but not always by a properly performed cystourethrogram, or by the gripping of an intra-urethral instrument. Clinically there may be a sudden interruption of the urinary stream followed by only partial bladder emptying; a significant bladder residual on catheterization immediately after voluntary urination; a normal cystometrogram and normal or reflex behavior of the bladder when an indwelling catheter has been left in place, together with a total inability of the patient to urinate as normally as these findings would indi

cate when the catheter is removed. Finally a sphincterogram will usually but *not always* yield final confirmatory evidence.

Sphincterometry. The purpose of a sphincterogram is to measure the resistance to the passage of a stream of fluid directed intra urethrally against the external face of the external urethral sphincter and at the same time to avoid all complicating artifacts such as leakage and so forth. In the male this is possible in most cases. In the female it is much more difficult. In both the bladder is emptied, if possible without the use of a catheter before making the test.

TECHNIC. The apparatus (Fig. 20) except for the mercury manometer is sterilized before use. It consists of a 2-ounce Asepto syringe the tip of which is inserted in the urethra. For use in the female this end is enlarged by pushing onto it an acorn-shaped flanged rubber diaphragm originally used to cover certain containers employed in the blood bank. This diaphragm is originally intact, but for use in sphincterometry its closed end must be pierced by a hot nail so that the nozzle of the Asepto syringe can penetrate it completely. This enlargement of the glass syringe tip is necessary in order to prevent back-flow from the urethra. The rubber diaphragm is known as the Fenwall Tel-O-Vac Diaphragm No. 9264 and is made by MacAllister and Bicknell. The other end of the Asepto syringe is closed with a perforated No. 4 rubber cork in which is placed one end of the horizontal arm of a glass T tube with an inside diameter of 4 mm. The other end of this horizontal arm is attached to the nozzle end of a 50-cc. syringe by an appropriate length of thick walled rubber tubing with an inside diameter of 3 mm. The vertical arm of the glass T tube is connected to one open end of a U-shaped mercury manometer with the scale marked in millimeters and with sufficient mercury in it so that with the manometer upright and level the meniscus in the open arm rests opposite the zero mark on the scale.

Before using the apparatus the operator must be certain that the plunger in the 50-cc. syringe moves very freely that the manometer connection is clamped off at its point of junction with the glass T tube and that all of the apparatus except the manometer and its connection is filled with a bland, sterile aqueous solution, such as normal salt or weak boric acid solution. The operator's assistant (this procedure cannot be carried out by one man alone) then inserts the tip of the Asepto syringe (with the rubber diaphragm in place if used in a female patient) into the urethra and compresses the latter with the fingers of his right hand in such a way as to produce as nearly as possible a watertight fit. With his left hand he supports the manometer in an upright position so that one meniscus of mercury is opposite the zero in the scale. The operator then removes the clamp between the T tube and the manometer and exerts pressure on the plunger of the 50-cc. syringe. This will produce a rise in the mercury in the U tube that will approximate in terms of millimeters of mercury the resistance offered by the external urethral sphincter to forcible passage of fluid from without inward. This figure is presumed to be the same in respect to the

forcible passage of fluid from within outward. The readings are only approximate and there is an element of error in the resiliency of the walls of the rubber connecting tubes and in the virtual impossibility of making the Asepto syringe-urethra joint watertight in either sex. Nevertheless, despite these possible inaccuracies the method has already more than justified its use.

Normal resistance of the external sphincter lies somewhere between 15 and 30 mm. of mercury. Readings of pressures higher than 30 mm. indicate abnormally increased resistance of the sphincter and hence the inescapable diagnosis of hypertonicity and spasm. It should be noted that the maximal pressure reading will register only momentarily on the manometer because just after this point has been reached the sphincter relaxes and the pressure of course falls to zero. An original pressure of zero in the apparatus, provided that everything is watertight and airtight, indicates an atonic external urethral sphincter that presumably is atonic because of denervation. Pressures between 0 and 15 mm. cannot be interpreted at present because of lack of experience and information.

Treatment. If there is sphincterometric and clinical evidence as noted above of a spastic or hypertonic external urethral sphincter and if the cord is not transected, a return to normal tonus with relief of these symptoms can be assured in most such patients by treatment directed toward temporarily destroying what seems to be a conditioned reflex. This permits the re-establishment of the normal sensory-stimulus and motor-response relation. This is permanent providing the circumstances that activated the abnormal conditioned reflex have been done away with. This desired result is accomplished by temporarily denervating one half of the sphincter at a time through the injection of a 1 per cent solution of procaine in the neighborhood of one internal pudic nerve where it crosses the ischial tuberosity as well as in Alcock's canal. The procedure is as follows. With the patient comfortably and firmly placed on one side with the lower leg straight and the upper flexed at the knee and hip so that the ischial tuberosity that is to be injected is uppermost and prominent, the tuberosity is located by palpation. The operator stands facing the back of the patient. If the patient has sensation in the area local anesthesia is used throughout; if there is anesthesia from the pre-existing disease or injury no other anesthetic is necessary. A three-inch short-bevel needle is then introduced inward, upward and laterally in such a direction as to strike the ischial tuberosity. A sufficient amount of a 1 per cent solution of procaine is injected in a fan-shaped area around this point with the purpose of anesthetizing the sensory branch of the internal pudic nerve as it crosses the tuberosity. The needle is then withdrawn and a longer one inserted through the same track, after which it is "walked" to the medial inferior edge of the tuberosity slipped over this edge and advanced laterally inward and upward underneath the shelf formed by the bone at which point more 1 per cent procaine is injected. If the injection has been made directly into the nerve in Alcock's canal, testicular pain will be caused. A fan-shaped area in this region is

anesthetized, care being taken *not* to direct the needle medially. Such a procedure would perforate the rectum. A properly placed injection will paralyze the sensory and motor branches of the internal pudic nerve and may cause a paralysis of one half of the external anal sphincter as evidenced by loss of the anal reflex on the side ipsilateral to the injection as well as certainly causing a paralysis of one half of the external urethral sphincter as evidenced by a significant reduction in the measurements obtained at a postinjection sphincterogram. Any amount of 1 per cent procaine solution (without Adrenalin) that is necessary up to 50 cc. may be used.

The peak of the effect of the injection will usually not appear for fifteen minutes and may not appear for thirty minutes. The anal sphincter will have regained its tone and reflex activity in twenty-four hours or less. The urethral sphincter will have regained normal tone in twelve hours or less, and spasms will not usually reappear for another forty-eight hours or more. Indeed, one injection may effect a permanent return to normalcy. A check sphincterogram and a determination of the residual urine at once after complete emptying without a catheter should be carried out the third day after the injection. Repeat injections may be made until it is apparent that relief of spasm has been effected or that the method has failed. Only one side should be injected at a time. In case of failure, careful scrutiny of the field for the basic activating original cause of the sphincteric spasm will nearly always prove that the former has been uncorrected and is still exerting its original harmful influence. The injection if successful also paralyzes a varying number and amount of the perineal muscles. This is merely a justifiable inference, however, since this paralysis cannot actually be demonstrated.

THE ATONIC OR FLACCID EXTERNAL URETHRAL SPHINCTER. This is the most disabling condition, as far as effective rehabilitation is concerned, that can affect any patient who has been paralyzed as the result of a spinal-cord or cauda-equina injury or as the result of a destructive injury of the pelvic wall or its contents. It predicates either complete bilateral destruction of the internal pudic nerves at any place in their peripheral course, complete bilateral division of the second, third and fourth sacral roots at any place from their point of origin in the sacral cord to the superficial face of the sacral intervertebral foramina by way of the cauda equina or a total destruction of the sacral spinal cord. *Treatment.* No effective direct method of attack on this problem has been devised. The inevitable spilling of whatever urine happens to be in the bladder whenever the patient stands erect can be cared for only by the use of pads, waterproof pants, the penile clamp and the male urinal. Catheters are useless, as the urine flows freely around the catheter. The only hope lies in the presence of an intact internal sphincter.

If a male patient who has not been circumcised decides to use a urinal constantly a circumcision should be performed. The redundant prepuce is certain to be excoriated and ulcerated by the constant wetting with urine in spite of the patient's best efforts at maintaining cleanliness, whenever a

rubber urinal of any semipermanent type is worn for long periods at a time. This is particularly true of the conventional male rubber urinal, which is worn strapped to the leg. This type has an "elephant's trunk" form of opening, into which the penis is thrust for its full length. Being made only in standard sizes, these urinals bear no necessary relation to the actual length or size of the penis to be inserted into them. Leakage and excoriation of the glans is inevitable. David Paull⁷ has evolved what appears to be a satisfactory way of getting around these difficulties (Fig. 21). The perforated



Fig. 21 David Paull's male urinal connection: condom, thick-walled small-bore rubber tubing; machined perforated plastic button; special skin cement.

closed end of a condom is stretched tightly over a plastic button with a hole in its center and is held in place by inserting the condom-covered button into one end of a thick-walled rubber tube with an inside diameter of 3 to 5 mm. This small diameter provides the siphon action that is so essential to the cleanliness of the penis. The other end of the rubber tubing is attached appropriately to the urinal *after removing* the "elephant's trunk." The condom is then slipped over the penis after the skin at the base of the shaft has been painted with "skin cement." The thin rubber of the condom makes an airtight connection with the skin. Since the other end of the condom-urinal connection is also airtight and since the small internal diameter of the rubber connecting tube creates a suction regardless of the patient's position, balanitis, excoriation, preputial swelling and so forth are eliminated. The condom is disposable and cheap. The skin cement is nonirritating. As a result, the penile connection can and should be changed three times a day with a soap-and-water cleansing done each time. Since a new condom is

used with each change further protection is given against excoriations caused by the otherwise necessary use of old apparatus. This method of controlling urinary incontinence traceable to a flaccid urethral sphincter is preferable to the use of a *Cunningham* (or *penile*) clamp. Since this clamp is essentially a spring clip that closes the urethra by exerting pressure through the shaft of the penis, it will cause pressure sores, edema, ischemic necrosis, ulceration and excoriations that may be difficult to cure unless the clamp is properly adjusted, frequently released, kept meticulously clean and used with intelligence.

Extravesicular Complications

THE OVERSIZED URETHRAL CATHETER. Perhaps the commonest complications incident to the proper care of the bladder in the presence of an injury to the spinal cord or cauda equina are those incident to the use of too large a urethral catheter. The indwelling catheter, whether used in the male or female—and particularly in the male—must never under any circumstances exceed 14 F. in size. The preferable type of catheter is whistle tipped or has the opening in the end. If such is not obtainable a rubber rectal tube of the same size is better than an ordinary side-opening catheter. The Foley type of catheter should not be used for prolonged periods. The bag irritates and in some cases has caused necrosis of the mucous membrane of the bladder wall when its adjustment has been disturbed. Its presence also tends to lead to overactivity of the detrusor. Any indwelling urethral catheter interferes with the normal drainage of the urethral secretions. If it is over 14 F. in size it may effectively preclude all possibility of such drainage.

URETHRITIS, EPIDIDYMITIS, ORCHITIS, SEMINAL VESICULITIS AND URETHRAL FISTULAS WITH EXTRAVASATION. The urethral glands and their secretions are always contaminated. If their discharge is blocked this contamination changes to infection and *urethritis* results. Following *urethritis*, *epididymitis*, *orchitis*, *seminal vesiculitis* and *urethral fistulas* with *extravasation of urine* succeed each other in rapid succession, present the usual signs and symptoms of infection in the particular organ involved, and should be referred to a genitourinary specialist for treatment. In the presence of *urethritis* the catheter should be removed and replaced with a new sterile one of proper size if catheter drainage is still possible; if this is not possible the catheter should be removed and not replaced until the local problem has cleared up. It is usually unnecessary to section and tie the vas deferens until after the third consecutive attack of *epididymitis* or *orchitis*. By the same token, if it is indicated, it is useless to postpone this operation at this time in the mistaken belief that the epididymis or even the testicle is still a functioning organ. Acute *epididymitis* and, more especially, *acute orchitis* should not be regarded lightly. A fatal *septicemia* may develop with death within twenty-four hours of the appearance of the first symptoms or signs of infection in the testicle. Urethral fistulas with or without *extravasation of urine* may be very difficult to close and the genitourinary sur-

geon should not be expected to undertake the closure of the chronic fistulas in the presence of involuntary spasms of the muscles of the legs abdomen or back.

COMPLETE DESTRUCTION OF THE URETHRA. This occurs in women who have insisted on disregarding the doctor's orders or have mistakenly been allowed to sit in a chair for too long a period before their nutritional state warrants it. This destruction will usually be only a part of a widespread neglected bed-sore problem, is commonly associated with bilateral ischial sores and may be complicated by osteomyelitis of the under surface of the symphysis of the pubis. The destroyed urethra will be replaced by a false passage between the bladder and the vagina with of course the total loss of all sphincteric control. There is no known treatment for this condition once it has been established. Any complicating osteomyelitis should be treated in accordance with well established methods and the associated bed sores dealt with as they would have been treated had the urethra remained intact. This is the type of case where waterproof dressings, made so by covering the ordinary dry sterile dressing with a piece of plastic sheeting held in place by adhesive strapping, will more than prove their worth. The dressings must be renewed daily or oftener and all weight bearing on the affected areas must be rigorously avoided.

STRICTURE OF THE URETHRA. The presence of a stricture of the urethra in the male may antedate the onset of the spinal-cord or cauda-equina injury. It is more likely however to result from the therapeutic use of urethral catheters that are too large, are not cleaned or are not removed and replaced often enough. The presence of a stricture greatly interferes with the rehabilitation of the bladder. Its treatment, therefore, should not be neglected. In particular the impassable type presents an extremely difficult problem from every point of view. In the major strictures therapy will undoubtedly include suprapubic drainage of the bladder and the presence of these strictures must be regarded as one of the definite indications for this operation in the spinal paralytic group of patients. This variety of stricture also has a bad genito-urinary tract prognosis, even with suprapubic drainage. The reason for this is that such bladder drainage in such patients inevitably leads to the formation of calculi, ureteral reflux, dilated ureters and hydronephrosis—any or all of which are nearly always complicated by infection, sooner or later. Some form of ureterostomy is usually the only practical method of saving any part of the urinary tract and may even have to be performed as a life-saving measure. Final decisions concerning types and methods of therapy however must be left to the genitourinary specialist.

THE HYPERTROPHIED PROSTATE. An injury of the central nervous system whether the spinal cord or cauda equina is involved or not, does not prevent the development of hypertrophy of the prostate or its enlargement from any other local cause. The obstruction to urinary outflow and associated bladder and other complications will be easily demonstrated if looked for. The treatment is the same as it would have been had there been no

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central-nervous-system injury and should be carried out by a competent genitourinary specialist.

Mechanical Complications

Certain complications that arise in connection with the evolution of a paralyzed bladder are primarily mechanical in nature and arise out of mis-conceived and misapplied methods of treatment.

SHRINKAGE AND FIBROSIS OF THE BLADDER WALL. This follows constant drainage that is used without interruption. It makes no difference whether it is the classic urethral constant drainage or drainage through a suprapubic, perineal or any other type of bladder or urethral fistula. The mechanical constant prevention of stretching of the detrusor leads first to contraction then to shrinkage and finally to fibrosis of that muscle. If infection in the bladder is added, the process will proceed with greater rapidity and a greater degree of permanent deficit. An unstretchable bladder that has a storage capacity of less than 30 cc. may become a permanent fixture in as short a time as six months of constant drainage to which has been added a low grade cystitis. The earliest stage of contraction is reversible if tidal drainage, with the apparatus properly set up and properly used, is resorted to at once (see page 84). The siphon loop must be set high enough to exert a stretching effect. Shrinkage can usually be greatly helped and perhaps completely corrected by the use of tidal drainage supplemented by repeated chemical partial denervation of the detrusor as described above (see page 104). A contracted, shrunken fibrosed bladder wall cannot be altered however, and as a result the patient is deprived of a reservoir for the collection of urine and cannot keep himself dry without the aid of a urinal used constantly day and night.

MECHANICAL OVERSTRETCH OF THE BLADDER WALL. This condition develops as a result of lack of understanding of the bladder physiology especially in connection with spinal shock. The naive beliefs that because there is an overflow from an overstretched overfilled atonic bladder it will come to no harm and that catheterization is unnecessary and therefore the bladder will take care of itself without help or if necessary can be aided in its efforts to prevent overdistention by manual forcing of its contents through strongly resistant sphincters lead to such physical damage to the fibers of the detrusor that they can only begin to shorten after the bladder is distended by 500 or 600 cc. of residual urine which never moves out of the bladder. Cystometrograms done on such bladders with the conventional fill of only 400 cc. (see page 89) give the appearance of an atonic detrusor. If the fill is continued until the mechanics of contraction again become possible any type of bladder function, from atonic to and including reflex, can be demonstrated. The best cure for this condition is prevention. Overflow retention, manual expression of urine, and the policy of no active therapy in the belief that the bladder can take care of itself should all be completely abandoned and cannot be condemned too strongly. Treatment otherwise is still in the formative stage. Talbot* has devised and performed an operation

on such mechanically permanently distended bladders that promises to correct an otherwise uncorrectable condition. A segment of bladder wall is removed and the cut surfaces resutured, thus rendering the mechanics of contraction more favorable for the remaining and now smaller detrusor.

URETERAL REFLUX. A complication of the overstretched bladder wall, whether on a mechanical or on a physiologically denervated basis, may be a ureteral reflux with an ascending ureteritis leading in turn to acute and chronic pyelitis, hydro-ureter, hydronephrosis and destruction of the kidney or kidneys. This condition develops because neglect of the bladder in its early stages or improper treatment (or both) has so stretched or damaged the bladder wall that the valvelike action, which under other conditions will prevent reflux of the contaminated or infected bladder urine up the ureter is done away with. The ureter and then the kidney is infected and then stretched by the transmission of the intravesical pressure upward, hydro-ureter and hydronephrosis develop and the function of the kidney is destroyed. Here again, the best cure is prevention. Early proper use of tidal drainage and of cystometrograms will eliminate all chance of ureteral reflux. If it and its complications are already present they must be treated as well as possible by a genitourinary specialist.

HYPERTROPHY OF THE INTERNAL URETHRAL SPHINCTER. Hypertrophy of the internal urethral sphincter is not related, except by chance, to spasm of the external urethral sphincter (see page 105). It is not a necessary accompaniment of the changes produced in the urinary bladder by injury to the spinal cord or cauda equina, as some surgeons would have us believe. Proper treatment of the bladder with tidal drainage from the very first day of the injury will prevent its development, barring other complications. Neglect in the early treatment, clinical and subclinical infections of the bladder and upper urinary tract, unnecessarily prolonged spinal shock and probably the use of too-large indwelling urethral catheters will lead to its development. I have not as yet seen it in women. The diagnosis is made by clinical evidence of obstruction of the emptying of the bladder in the absence of calculi, hypertrophied prostate or spasm of the external urethral sphincter plus the cystoscopic findings and demonstration of an appropriate deformity by a cystourethrogram. The first and last criteria give the more accurate and reliable data for diagnosis. Treatment should be in the hands of a genitourinary surgeon; he should never hesitate to remove too little tissue rather than too much. I have seen patients who were continent and rehabilitatable as long as they were willing to put up with an indwelling catheter rendered permanently incontinent—catheter or not—and permanently unrehabilitatable following an excessive so-called resection of the internal urethral sphincter. This is a tragedy that must be avoided at all costs.

After the obstructing tissue is removed the hypertonic detrusor must be dealt with. This may be possible through the proper use of tidal drainage. If it is not, the sacral nerves should be injected as described on page 104.

Failure to recognize this need will obviate any success that might have been obtained by the resection of the tissue

Complications Based on Infection

ACUTE CYSTITIS Acute cystitis is a preventable infection and should never occur. It is always a potential danger, however, as long as an indwelling catheter must remain in place. It has been conclusively demonstrated that in the absence of any other cause the presence of a sterile, small, indwelling urethral catheter that is in place in either male or female for seventy-two hours or longer is *always* associated with bacterial contamination of urine. In the absence of bladder stones and residual urine and with the aid of tidal drainage, this contamination will remain contamination and not become an infection. If residual urine collects, if stones form or are deposited from the kidneys, if the tidal-drainage apparatus is improperly set up and used, if cystometrograms have been too infrequent, if the catheter is too large, becomes encrusted or is not changed every week, if the mucous membrane is irritated by the use of improper irrigating solutions, if a suprapubic or perineal bladder or urethral fistula is present or if the bladder has become either overcontracted or overdistended through neglect, a subacute or acute cystitis will develop. It is best treated by forcing fluids—preferably water and preferably by mouth—up to 5000 cc. per twenty-four hours if the patient's kidneys and heart are normal and he or she is an adult. The activating factors, some of which are listed above, must be corrected also. Additional therapy, whether drugs or not, is of little moment one way or the other. Usually, however, although not actually helpful, it is at least harmless, unless it leads to the neglect of the all-important correction of the activating element and the administration of sufficient fluid.

For the treatment of acute urethritis, acute epididymitis, acute orchitis and acute seminal vesiculitis, see page 192.

ACUTE URETERITIS. This results from an extension of an infection that is present in the bladder or in the renal pelvis or from a block of the urinary flow by a stone, stricture, scar or the like. Treatment should be in the hands of a genitourinary specialist.

ACUTE PYELITIS. In spinal-cord and cauda-equina injuries, infection in the renal pelvis will have ascended from the bladder. It will not develop, except in rare instances, if the patient and his bladder are adequately cared for by properly used tidal drainage. When it is present, its therapy is a problem for the genitourinary specialist.

PYELONEPHROSIS, CARBUNCLE OF THE KIDNEY, PURULENT OBSTRUCTIVE PYELITIS. All these conditions, when they develop as complications of spinal-cord and cauda-equina injuries, are preventable by appropriate and proper treatment of the genitourinary tract from the very start. They are unnecessary complications of the central-nervous-system injury. Their treatment should be in the hands of a competent genitourinary specialist.

RENAL AND BLADDER STONES. It is apparent that renal and bladder stones are significant complications of spinal-cord and cauda-equina injuries only when early treatment of the genitourinary tract has been improper neglected or absent. As a result, the genitourinary tract has become infected from top to toe, the patient has had to be immobilized more completely and for longer periods of time than would otherwise have been necessary and the stage has been set for the formation of the first stones. If they are in the bladder alone, and if they are removed promptly and treatment otherwise is revised bladder obstructions eliminated and the patient mobilized, they may not re-form. If the catheter can be eliminated as well the chance of their return is diminished still further. If they are in the kidney alone or in both kidneys and the bladder their very presence perpetuates the clinical and frequently subclinical infection that activated and maintains them, and they will re-form again and again until the infection and every last stone have been eliminated. The chance of doing this, once the process has begun is very poor. This is one condition that virtually can be only eliminated by prevention. If that has been neglected and stones are already present, their treatment should be in the hands of a genitourinary specialist. Only properly used tidal drainage from the very start, the ingestion of at least 3600 cc. of water per twenty four hours, a proper diet with enough calories and protein and early bed mobilization are effective as preventive measures. Special diets and other similar methods are not needed and are not effective.

Complications Produced by Surgery

SUPRAPUBIC CYSTOSTOMY The use of this surgical procedure as a method of caring for the bladder that is disabled on account of a spinal-cord or cauda-equina injury is never indicated unless the purpose of the operation is to remove a stone or foreign body from the bladder or to supplement the surgical closure of a urethral fistula. In the first two instances it should be closed at the end of the operation and bladder emptying cared for by an indwelling urethral catheter. Experience has demonstrated that suprapubic cystostomy is even contraindicated under combat conditions. The contraindication holds whether the drainage is provided by a formal operative incision through the bladder wall or is accomplished through a suprapubic stab wound with a trocar. Such drainage leads to shrinkage of the bladder wall, infections of its cavity the collection of residual urine, the formation of bladder stones, prolonged unnecessary functional disability of the bladder, maceration of the skin, more frequent formation of bed sores, unnecessary pain, prolongation of spinal shock, increased difficulty of rehabilitation, unnecessary surgery and increased morbidity and mortality.

PERINEAL CYSTOSTOMY This is mentioned only in order to condemn it. It has no place in the treatment of these bladders. In addition to the complications listed above under *Suprapubic Cystostomy* it presents the added difficulty of a urethral stricture and is an impediment to bladder drainage when the patient sits erect and thus kinks or compresses the drainage tube.

Constant Urethral Drainage with or without Manual Irrigation

Constant urethral catheter drainage alone is inefficient, leaves residual bladder urine and promotes unnecessary bladder and higher urinary-tract infection and shrinkage of the bladder capacity. It offers no advantage over tidal drainage and presents many disadvantages. When combined with regular intermittent manual irrigation, it is more useful than without that addition but is preferable to tidal drainage only when the bladder has collected such large amounts of mucus, blood, crystals or small stones that the urethral catheter cannot otherwise be kept patent. Solutions M or G of Suby et al.⁶ 1:20,000 aqueous Zephiran or 1/2 of 1 per cent acetic acid solutions make the most useful irrigating fluids. Potassium permanganate, boric acid, tap water and normal salt solutions should not be used, as their use promotes the conditions described above as necessitating manual irrigation. If constant drainage with manual irrigation is indicated the drainage itself should be intermittent in order that the intervening filling of the bladder may serve to keep its walls stretched. This method is the next preferable one to tidal drainage but nevertheless should never be used except where specifically indicated as listed above.

The Syndrome of Sympathetic Nervous System Overactivity and Visceral Distention

This syndrome is most commonly seen in transecting cord injuries and in relation to distention of the urinary bladder although distention of the large bowel may also cause it. It has been noted in patients with cord injuries at all levels but never in the presence of uncomplicated cauda-equina injuries. It is impossible to predict in advance what patient will and what patient will not show the syndrome. It does not occur during the period of spinal shock and disappears after it has manifested itself with the redevelopment of spinal shock from whatever cause, only to recur when the spinal shock has disappeared in its turn. It is more common with the hyper-tonic reflex and reflex bladders than with any other type but may also occur with distention of the large bowel. In a typical attack, the bladder contents reach a critical level of fill, which varies with each patient. When this level is passed, further fill and resulting bladder distention cause profuse sweating, usually but not invariably or exclusively in the paralyzed area of the body. This is succeeded, if the bladder is not emptied at once by a severe pounding headache. If the process is allowed to continue, the blood pressure rises—perhaps to very high figures—and unconsciousness develops, with or without convulsive seizures, which, it is said, have been followed by death in certain not too well authenticated instances. The attack is aborted if the bladder or large bowel, as the case may be, is emptied prior to unconsciousness. The attacks can be prevented by constantly restricting the bladder fill to less than the critical level. This will not cure the attacks, however and since their presence so limits the reservoir capacity of the bladder and bowel as to prevent adequate bladder and bowel training, they may effectively prevent what would otherwise be a satisfactory rehabilitation.

The mechanism and physiopathology of the attacks are not well understood, but it is probable that some form of rather widespread reflex disturbance of the autonomic system function is set up. Drugs that affect the sympathetic nervous system have been administered without any constant effect. Injection of the sacral roots in the same manner that has been described for the treatment of a hypertonic detrusor (see page 104) and injection of the internal pudic nerves as used in the treatment of a spastic external urethral sphincter (see page 105) have given relief, but the number of cases that have been subjected to this therapy is too small as yet to promise anything definite. Mobilization has seemed to help at times, and at times the attacks disappear of their own accord and for no explainable reason.

Drugs in the Control of Urinary Infection

My experience with the use of drugs to control the contamination and possible infection of the urinary tract in patients with spinal-cord and cauda-equina injuries has been disappointing. They are not necessary if the bacteria are held to a contamination level because removal of the foreign body whether catheter or calculus, will permit the return of the urine to its relatively sterile normal state. Water preferably given by mouth at the rate of 5000 cc. per twenty-four hours for an otherwise normal adult, has done more in my hands, to control genitourinary-tract infection than any type or amount of drug—provided, however that tidal drainage is properly used in conjunction with the water and provided further that in infections external to the bladder the urethral catheter is withdrawn at once and left out until the infection has subsided. Of all the drugs streptomycin seems to be the most useful but at times penicillin Chloromycetin Aureomycin, Gantrisin and the various sulfonamides have been effective. It is definitely useful to keep the bladder urine on the acid side. In these cases this is most efficiently done by using $\frac{1}{2}$ of 1 per cent acetic acid solution as the irrigating solution in the tidal drainage apparatus. Potassium permanganate is dangerous because it is actually alkaline. It should never be used as an irrigating solution. No matter what therapy is used however it is impossible to permanently control infection while stones are present anywhere in the genitourinary tract, and it is impossible to prevent contamination from which infection gets its start under adverse conditions of treatment, as long as an indwelling catheter must be used.

Osteoporosis

The metabolism of calcium particularly in its relation to osteoporosis of the bones of the paralyzed portion of the body and extremities, has barely been scratched, and much more needs to be learned than has been learned to date. The following are among the limited facts now available to us, and appear to be true. The osteoporosis that appears in connection with injuries of the spinal cord and cauda equina is limited to the bony structures of the

weight-bearing mobilization must be guarded in the face of clinical osteoporosis, especially when mobilization has been accompanied by or has caused multiple fractures of the bones of the paralyzed extremities.

Sexual Function

Sexual Function in Injuries to the Spinal Cord and Cauda Equina in Men

In men this function must be considered from the two points of view of fertility and potency

FERTILITY The best evidence indicates that traumatic injury to the spinal cord and cauda equina does not preclude fertility. On the other hand, such common unnecessary complications as repeated attacks of epididymitis or orchitis (see page 111) and surgical division of the vas deferens may cause it. Before final decision is or can be reached concerning the fertility of the individual male, therefore, testicular biopsies, sperm counts and other studies on the semen should be carried out by a specialist in this field.

POTENCY ¹⁹ Potency depends on the ability of the subject to have an erection as well as an ejaculation and to accomplish intromission.

Erection takes place on a purely segmental reflex basis. Suprasegmental connections are not necessary. The reflex may be activated by either general psychic or local tactile stimuli. It is to be assumed that the former implies some degree of integrity of the pathways that connect the spinal reflex centers and the suprasegmental areas. Their location is unknown but appears to be in the spinal cord, since complete transection of the cord at any level abolishes their transmission. Tactile stimulation of the glans sets up sensory impulses that travel to the second, third and fourth sacral segments by way of the internal pudic nerves. Efferent impulses leave these same segments as a reflex response to these sensory stimuli, travel by way of the parasympathetic nerves and cause dilatation of the arterioles of the penis, with distention and congestion of the corpora cavernosa and corpus spongiosum. Other efferent impulses, stimulated by the same sensory mechanism, leave these same segments simultaneously over the internal pudic nerves to cause contraction of the penourethral muscles with resultant compression of the venous drainage channels of the penis. The only anatomic conditions that will prevent erection are complete destruction of the sacral spinal segments or their roots or both, transection and destruction of the cauda equina and transection and destruction of all the pelvic parasympathetic plexuses. In man there seems to be no "center" for erection in the spinal cord. This reflex act can be completely inhibited or abolished, however, by psychic impulses. If such psychic inhibition can be controlled, all male patients that have been subjected to a spinal-cord or cauda-equina injury regardless of level or type except as noted above have the anatomic and neurologic equipment to have an erection. All otherwise normal male patients, whether they can have an erection or not, will retain their libidinous interest in the opposite sex.

Ejaculation ²⁰ takes place so far as our present evidence indicates, as the result of a complex purely spinal segmental reflex. I do not believe that

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there is a so-called spinal center for ejaculation and I think that the findings in animals to that effect by Kuntz¹¹ and others do not hold true for men. In any event it is certain that suprasegmental connections are not essential for the act that once the reflex mechanism has been put in motion it cannot be inhibited that the act cannot be initiated voluntarily and that it must be preceded by and take place during an erection.

Ejaculation occurs as the result of neural stimulation by way of two reflex arcs. The original sensory stimulus is formed by a summation of impulses generated in the spinal cord during the process of erection and accompanied in normal persons by stimulation of the glans. When this summation reaches a threshold level, there is a sudden discharge of efferent impulses. These produce a sudden contraction of the smooth muscles of the entire sexual apparatus with resultant propulsion of the semen into the urethra. The afferent stimuli of this first reflex travel toward the second, third and fourth sacral spinal segments over the internal pudic nerves. The efferent or motor impulses which respond reflexly to the sensory stimuli, travel over the sympathetic nervous system reaching the peripheral part of this network by connections to it from the spinal cord between the sixth thoracic and fourth lumbar segments. The presence of semen in the urethra then acts as a sensory stimulus to activate a second reflex act. Both afferent and efferent impulses that are part of this second reflex travel over the internal pudic nerves to and from the second, third and fourth sacral and upper lumbar spinal segments. They cause contraction of the constrictor urethri bulbocavernosus and ischiocavernosus muscles. These contractions expel the semen from the meatus. During the ejaculatory act the prostate is stimulated its secretion is added to the semen in the prostatic urethra, and the internal vesical sphincter contracts blocking off the bladder. Once initiated this part of the reflex act cannot be prevented. On the other hand it cannot be voluntarily initiated, either. The whole act of ejaculation is dependent on a pre-existing erection and can be prevented only indirectly and by the inhibition of that erection. The only anatomic conditions that will prevent erection, in addition to those that do away with the necessary preliminary or both a transection or destructive lesion of the cauda equina, spinal-cord damage between the sixth thoracic and third lumbar segments so severe and so widespread as virtually to destroy the sympathetic components of the cord in this area, and complete detachment of the sympathetic connections from the cord between the eleventh thoracic and first sacral segments.

The Orgasm Sexual orgasm is associated with ejaculation and constitutes the sensations caused thereby. It is not known how or where these sensations arise but it need not be assumed that they reach the cerebral cortex. They do however give rise to reactions in other viscera. The excitation apparently spreads through the autonomic nervous system. The sensations also give rise to somatic reflexes in addition to those activating the perineural muscles and thus produce spastic contractions of the lower extremities, which are usually supplemented by voluntary contractions.

of other muscles throughout the body. The sensations of pleasure and their appreciation that accompany the orgasm, on the other hand, require intact central connections. These sensations are absent in patients whose cord is transected above the sacral segmental level.

Intromission Effective coitus may be barred by the physical impossibility of effecting intromission despite the presence of an erection. This disability will arise from the somatic paralysis of the legs and trunk. Despite the best efforts of both parties, nothing can be accomplished as long as the paralyzed male remains the aggressor. In such instances successful coitus and resulting impregnation can be accomplished by reversing the physical relationship of the individuals and at the same time depending upon the female for any necessary aggression.

Sexual Function in Injuries to the Spinal Cord and Cauda Equina in Women

Much less is known in detail about the effect of spinal-cord and cauda-equina injuries on the reproductive and sexual activity of women than is known about its effect on these activities in men. It is to be expected that the neural mechanism will conform in all but certain details to that known to function in men, and, indeed, this appears to be true. Kuntz¹¹ points out that the ovary and the fallopian tubes are innervated by branches from the sympathetic plexuses (especially the hypogastric plexus) and by other branches that travel with blood vessels. The central connections are through the tenth, eleventh and twelfth thoracic nerves and the lumbar nerves. The uterus and vagina receive innervation from both sympathetic and parasympathetic origins, the nerves arising from the uterovaginal plexus. This corresponds to the prostatic plexus in the male. It is continuous superiorly with the hypogastric plexus. Other sympathetic fibers come from the lower lumbar and sacral sympathetic trunk. The parasympathetic fibers originate in the sacral spinal cord and travel by way of the pelvic nerves. The clitoris is surrounded by a cavernous plexus with both sympathetic and parasympathetic fibers that are closely associated with the vascular supply to the organ and are also connected with the somatic nervous system by way of a branch from each of the internal pudic nerves. The labia have their chief neural connections with the somatic nerves by way of the ilio-inguinal, the internal pudic and the perineal branch of the posterior cutaneous nerve of the thigh. The autonomic supply comes from the vesical and vaginal plexuses.

MENSTRUATION OVULATION INTRA-UTERINE GROWTH OF THE FETUS AND NORMAL DELIVERY Kuntz further states that "there is no conclusive evidence for the direct functional innervation either of the ovarian follicles or the interstitial secretory tissue; therefore it cannot be assumed that the ovarian functions are subject to direct nervous regulation." If this holds true, there should be no interference with either *menstruation* or *ovulation*, so far as the ovarian function is concerned, by any level or type of injury to the spinal cord or cauda equina. This is known to be so. He further points

out that the "smooth musculature of the female genital tract has the capacity to undergo rhythmic contractions in the absence of nerve impulses. The activity of the musculature is regulated in part through hormonal agents and in part through its innervation." Thus normal *intra-uterine growth of the fetus*—other things being equal—as well as *normal delivery* at term should take place in the presence of any type or level of injury to the spinal cord or cauda equina. It is known that this is also true.

GENITAL REFLEXES. The control of genital reflexes is said to be identical in the two sexes and to be located in the sacral and thoracolumbar segments of the cord in women. The same is true of the neural pathways over which the respective sensory and motor impulses, whether autonomic or somatic, travel. Thus, stimulation of the clitoris produces engorgement and erection of this organ, which is analogous to the erection in the male and culminates in a summation of sensory impulses arising in the external genitalia. A discharge of efferent impulses over the thoracolumbar sympathetic connections follows and induces emptying of Bartholin's glands, a discharge that is analogous to the expulsion of seminal fluid in the male. At the same time rhythmic peristaltic contractions that are propagated from the fallopian tubes to the uterine musculature and that cause expulsion of mucus will be caused by neural impulses from the "summation pool." At the height of sexual excitement these contractions culminate (after spreading to the vagina) in rhythmic contractions of the sphincteric vaginal muscles which are activated by somatic nerves.

ORGASM. Orgasm, which, as in the male is a sensory appreciation of the culmination of the sex act, requires central suprasegmental connections in both sexes. It is therefore absent in both sexes whenever the spinal cord is transected. The neural pathways involved appear to be the same in women as those in men. Destruction of the sacral cord or roots, or of the lower thoracic and upper lumbar cord or its connections with the sympathetic paravertebral ganglionated chain, or destruction of the cauda equina, should prevent the formation of a favorable mechanical atmosphere for the impregnation of any ovum that has been made available despite the neurologic deficit. Sufficient experience has not accumulated as yet to permit a dogmatic statement in this regard.

In summary it is known that menstruation, ovulation, coitus, *impregnation* (so far as the nonmechanical factors are concerned) *intra-uterine fetal growth*, normal delivery and the normal development of the breasts with normal lactation can take place if not interfered with by any other cause. In spite of the presence of any level, type or degree of injury to the spinal cord or cauda equina. It is further known that orgasm in the female will be absent if the spinal cord is transected above the sacral segmental level. It is assumed that destructive lesions of the sacral cord, its roots, the cauda equina and the thoracolumbar cord or its connections with the paravertebral sympathetic ganglionated chain will in some measure interfere with the genital reflexes and the sexual gratification that is connected with coitus.

The Effect of Anterior Dorsolumbar Rhizotomy on Sexual Activity

In men a properly performed anterior dorsolumbar rhizotomy will not prevent erection and will not preclude fertility. This does not mean that erections that were present prior to the rhizotomy may not be lost after that operation. It does mean, however, that in all such cases that have been studied the loss of erection could have been caused by any one of a number of possible alternatives. There are as yet not enough data to be certain about the effect of rhizotomy on ejaculation. Tentatively on anatomic grounds, it appears most reasonable to assume that unless the operation is done on a patient who has sustained a terrifically widely destructive injury of the thoracolumbar cord the operation will have no effect on the ejaculation.

In women the operation has been shown to have no effect on menstruation, ovulation, impregnation, normal development and normal delivery of the fetus.

Care of the Bowel*Normal Defecation*^{12, 13}

The process of normal defecation depends first on the regulated peristaltic passage of the food from the upper end of the esophagus to the descending colon, secondly upon its storage and solidification in the descending colon and sigmoid, and finally on its intermittent delivery to the otherwise empty rectum for discharge through the anal canal. This procedure is controlled to approximately the beginning of the jejunum, first by the somatic nerves of the mouth and throat and then by the thoracic parasympathetic nerve supply. From this point to the descending colon the sympathetic plexuses on the posterior abdominal wall take over. They are succeeded by the parasympathetic plexuses of the pelvis which act on the descending colon, the sigmoid, the rectum and the internal anal sphincter. Finally the somatic nerves supply the perineal muscles and the external anal sphincter. The parasympathetic and sympathetic neurones are everywhere intermingled, and all parts of the autonomic system extend to all parts of the gastrointestinal tract. The supply of one part, however, roughly predominates in certain areas as indicated above. Correlation and orderly transmission of these motor impulses are governed by the intramural plexuses of Meissner and Auerbach and thereby produce peristalsis. These latter plexuses extend the full length of the intestinal tract. Chemical, hormonal and glandular products also govern the mechanical mixing, the digestion, the absorption and degree of hydration of the food bolus and, secondarily the rate of its progress through the tract. When the bolus has been reduced to waste products only and is ready for excretion, it has reached and is stored in the descending colon and sigmoid.

Inhibition of peristalsis, the creation of bottlenecks by spasm, interference with the orderly chemical and hormonal digestive processes and also reverse peristalsis may be caused by psychic influences. This is but an extension of the normal inhibitory influence that may be brought to bear on the empty

ing of the bowel by impulses arising in the higher centers. The seat of origin of these impulses and the points of their impact on the central or autonomic nervous system are not known. There can be no doubt about their effect, however. Except by direct stimulation of the intestinal walls, either through the abdominal wall or by digital insertion into the anal canal, it is impossible for the normal person to initiate peristalsis of either the small or the large bowel.

The act of normal defecation therefore concerns the descending colon, the sigmoid, the rectum, the internal and external anal sphincters, the perineal musculature and the anal canal. As detailed by Starling¹² in a description that has not been superseded, it is usually initiated by a voluntary increase of the intra-abdominal pressure. To accomplish this, the patient holds his breath and compresses the anterior abdominal muscles. In response to this stimulation, part of the stored contents of the sigmoid is moved into the hitherto empty rectum. This shift of contents may also take place without voluntary contraction of the anterior abdominal muscles but rather by peristalsis alone. Its place is taken in the sigmoid by material advanced from above from the descending colon. The contents of the rectum are thus increased until there is sufficient stretch of its walls to initiate a sensory stimulus, which promulgates a peristaltic wave that in its turn ends with a simultaneous contraction of the rectal wall and relaxation of the internal anal sphincter. This activity is apparent to everyone who has ever passed flatus unexpectedly and inappropriately. This particular sensorimotor reflex is mediated through the parasympathetic pelvic nerves and the second, third and fourth sacral spinal segments. With the relaxation of the internal sphincter some of the fecal content moves from the rectum into the intersphincteric portion of the anal canal and there sets up a second sensorimotor reflex. The impulses that mediate this second reflex travel in both directions over the somatic internal pudic nerves and produce relaxation of the external anal sphincter, contraction of the perineal muscles and elevation of the levator ani muscles. This combined activity opens the anal passageway, permits the discharge of the rectal contents and straightens out the recto-anal canal. The rectum empties itself completely during this succession of events. If fecal content is still left in the sigmoid, and possibly also in the descending colon, this remaining material is moved to the rectum from which it is expelled through the anal sphincters and canal by a repetition of the same process. Except during defecation the rectum normally remains empty. Stimulation adequate to produce a rectal emptying contraction can be provided by either gas or solid material. Liquid material in the sigmoid or rectum, however, especially if irritating, will cause hyperperistalsis, with waves starting high up in the colon and, in severe cases, rectal tenesmus. Ideally, normal defecation takes place as a conditioned reflex and is most efficient if no effort or conscious control is exerted. In particular, conscious attempts to hurry the process lead to inhibition and thus to interference with this conditioned reflex activity. Defecation should take place in a warm

room on a comfortable toilet seat, at leisure and at the same time every day. It will be facilitated if the individual reads, smokes a cigarette or otherwise promotes mental relaxation and inattention to the movement of his bowels.

Bowel Care in Association with Injuries of the Spinal Cord and Cauda Equina

Care of the bowel in patients suffering from the results of an injury to the spinal cord or cauda equina is, for practical purposes, limited to those patients in whom the bowel has been deprived by the injury of its cerebral connections or has been denervated by destruction of the sacral cord or its roots or of the peripheral nerves formed from them. Such deficiencies imply a transection or near-transection of the cord at any level, destruction of the sacral portion of the cord or its roots or destruction of the parasympathetic supply and the external pudic nerves in the pelvis. This has been dealt with in general under the appropriate headings in the section *Injuries to the Spinal Cord and Cauda Equina* (see page 56). This section will therefore deal only with *bowel training* and certain *miscellaneous associated problems*.

BOWEL TRAINING This is a necessary prerequisite for complete rehabilitation in those patients who have had a cord transection. Success in bowel training depends on the formation in the patient of a conditioned reflex that, when invoked, will cause prompt and total emptying of the rectum and ascending colon once every twenty-four or forty-eight hours. This training will virtually guarantee that the bowel will not discharge its contents at any time other than the chosen one provided that no local irritation is allowed to develop. To facilitate such regularity the contents must be kept in such condition, by an appropriate diet, that the stool is soft and formed when it reaches the rectum.

For initiation of bowel training the patient must be free of spinal shock, able to sit erect, and not constipated. He must have an understanding of the problem to be solved and he must be willing to make the necessary co-operative effort to attain that solution. There must be sufficient remaining neural connections, both autonomic and somatic, to assure the maintenance of those reflex arcs that mediate the peristalsis of the descending colon and rectum and the contraction and relaxation of both sphincters. As a starting point, the patient must choose some specific time of day at which he proposes to empty his bowel for the rest of his life. Each day at that time, he is given an enema and then seated upright on a bed pan or preferably a commode, if his mobilization has not reached the point at which he can sit on the toilet. If his mobility and training permit, however, he should take the enema in the bathroom just before sitting on the toilet. After approximately a week of this program, the enemas are gradually decreased in amount over another week or ten days. At this time the enemas should be omitted on every other day and a small dose of milk of magnesia given. This must be ingested at such a time prior to going to stool as to ensure that its cathartic effect coincides with the time chosen for emptying the bowel. The proper time and dose of milk of magnesia must be determined by ex-

periment. At this same time the patient is started on mineral oil by mouth—1 ounce—night and morning. This dose is continued until the oil begins to leak from the anus, after which it is reduced to a point at which it keeps the stool soft but still formed and no longer leaks between defecations. The enemas are now gradually eliminated, and the use of milk of magnesia is increased in frequency sufficient to ensure daily or bldaily movements. After the enemas have been eliminated the milk of magnesia is then eliminated in its turn by reduction of the amount of each individual dose and by reducing the frequency of the doses until it too is no longer necessary. At this point the stimulation provided by the patient's being in position to defecate will be enough to ensure its taking place. It is not necessary to strongly compress the abdomen as an aid to defecation under these circumstances—indeed, it is not desirable. However gentle rotary rubbing of the anterior abdominal wall simultaneously with the expected activation of the bowel first by the enemas and then by the milk of magnesia is helpful and desirable during the training period. Intra-anal digital stimulation prior to defecation should not be necessary. Harsh cathartics and, indeed, any cathartics except mineral oil by mouth should never be used except for definite indications. Under such circumstances milk of magnesia should be used again. Some patients do better if the bulk of their stools is increased by the use of a bland agar preparation. This can best be determined by experiment. Active ambulation is of the greatest help in maintaining regularity of bowel movements by such a conditioned reflex mechanism.

Bowel Training in Patients with Destructive Injuries of the Sacral Spinal Cord or Its Roots or Both. The usual injury in such patients does not completely divide the parasympathetic supply to the large bowel. For this reason the colonic and rectal peristalsis is still active and the internal anal sphincter continues to act reflexly even in the face of a flaccid or atonic external sphincter. Such patients are continent and can be trained to move their bowels by institution of a conditioned reflex, as described in the previous section.

Bowel Training in Patients with Complete Denervation of the Pelvic Colon Rectum and Sphincters. This condition results from complete destruction of the cauda equina or extensive injuries to the pelvis and its walls with bilateral complete obliteration of the parasympathetic plexuses and both internal pudic nerves. These patients are not and cannot be continent according to presently available knowledge. Bowel training is impossible. Their best hope is a well placed, properly constructed colostomy to which is fitted a properly constructed colostomy bag. The patients must be trained in the proper handling of the colostomy. This training and the construction of the colostomy should be turned over to a general surgeon skilled in large bowel surgery.

Bowel Training in Association with Cranio-cerebral Injuries. No particular problem is associated with care of the bowel in the presence of a cranio-cerebral injury. The standard enemas and cathartics are used in usual ways. The operator should bear in mind, however that saline

tics and particularly magnesium sulfate used as a purge are equally effective as dehydrators and unless he wishes to produce a double effect it is better to use some other variety of cathartic and one that is not of itself dehydrating. It should also not be forgotten that violent purging, no matter what the means used is also extremely dehydrating.

Miscellaneous Data

ANAL REFLEX. Integrity of the somatic nerve supply to the external anal sphincter can be determined by demonstration of the anal reflex and by the presence of a palpable contraction of any part of this muscle on the finger inserted into the anal canal.

IMPACTIONS Impactions must be carefully guarded against at all times in patients with spinal-cord and cauda-equina injuries, even after bowel control has been attained. The presence of an impaction is usually manifested by unexplained intermittent attacks of diarrhea. The discharged bowel contents will contain a high percentage of mucus, but will reveal no evidence of blood. If the impaction cannot be removed manually it should be softened first by an oil-retention enema, which is then followed by a milk and molasses enema.

THE INTEGRITY OF THE PARASYMPATHETIC NERVE SUPPLY to the descending colon, sigmoid and rectum with its internal sphincter can most easily be demonstrated by a study of a cystometrogram. An atonic bladder and external urethral sphincter will almost always indicate a totally denervated rectum and anal canal unless the atonicity has been caused by the presence of spinal shock. An autonomous bladder predicates a partially denervated rectum and anal canal a reflex bladder a rectum and anal canal that have a normal segmental nerve supply.

Turning a Patient in Bed with a Draw Sheet

The use of the ordinary linen draw sheet that is put on every hospital bed to cover the rubber draw sheet is the best, most efficient and least dangerous way to turn a patient who is confined to bed as the result of a spinal-cord or cauda-equina injury. Only in this way is it possible to move the entire body and head as a unit and thus avoid adding to the damage already present. Since all such patients must be moved from side to back and then to the other side again every two hours while they are bedridden, it is important to have such a trouble-free method available. Of course, the same thing, so far as moving is concerned, can be accomplished by using the Stryker frame or one of its modifications, but this cumbersome apparatus is expensive, is no more efficient and has such other contraindications that I have yet to find its use advisable.

When the patient is to be moved, the edges of the draw sheet are pulled out from beneath the mattress on both sides and rolled up until they can be grasped firmly. One or more attendants, according to the size and weight of the patient, grasp the rolled edges of the sheet on either side of the patient. If a cervical injury is present another attendant must hold the sides

of the patient's head and turn it and the neck at the same rate as the body is turned. Only in this way will flexion, torsion or lateral bending of the cervical spine be prevented. The opposite attendants pull against each other and tighten the sheet under the patient (Fig. 22). These maneuvers are preliminary to the actual turning, which is carried out as follows. To turn the patient from his back to his left side for example, the attendant or attendants on the patient's right maintain their pull on the draw sheet and



Figs. 22 through 25 Turning a patient with a cervical cord injury to his left side with the aid of the draw sheet. Fig. 22. The draw sheet pulled tight beneath the patient.

in addition lift its edge up and toward the patient's left side. During this procedure the attendant or attendants at the patient's left merely keep the sheet tight (Fig. 23). This rotates the patient in one piece (Fig. 24) but places him dangerously close to the left edge of the bed. To correct this, both sets of attendants maintain their hands and the sheet in the same relative positions and acting in concert lift the patient without changing his newly rotated position and move him bodily to the center of the bed (Fig. 25). With the patient thus properly placed, and after his legs, body and arms are adjusted, protected and stabilized by pillows, the sheet is again tucked beneath the mattress and the covers are readjusted. To turn the patient from his back to his right side the previous steps are carried out with the sides reversed.

To change a patient from his side to his back, he is lifted on the draw sheet, after it has been tightened, and moved to the side of the bed away from which he is to be turned. After straightening the patient's legs, the attendant facing him holds the sheet tight and horizontal. The attendant behind the patient brings his part of the sheet against the patient's back,



Fig. 23 The patient rolled toward his left.

after which the two attendants, working in concert, turn him toward his back while supporting him front and back with the sheet, until he is flat in the middle of the bed. There, after his legs, body and arms are adjusted, protected and stabilized by pillows, the draw sheet is again tucked beneath the mattress and the covers are readjusted.

After this has been demonstrated and practiced once or twice, this method of turning patients can be easily carried out by two nurses or orderlies (three in the cervical cases) except when the patient is excessively large or heavy when four or five will be necessary. No other method is safe or can be countenanced, no matter what the level of injury. Neither hyperextension on a blanket roll nor traction with Crutchfield tongs or a flannel-bandage halter contraindicates the use of this method.

Braces and Splinting

No part of any patient with a spinal-cord or cauda-equina injury should ever be enclosed in plaster of paris

The need for braces and splinting of various parts of the body in patients with spinal-cord and cauda-equina injuries falls into four categories. The



Fig. 24 The patient turned on his left side at the edge of the bed.

first has to do with emergency splinting at the scene of the accident the others concern any other splinting that is necessary until the vertebral injury has been healed and is permanently stabilized in position and the patient is as fully ambulant as his injury will permit.

Emergency Splinting

The one particular emergency in cases of spinal-cord injuries that require immediate and adequate splinting, is the cervical injury. Here the danger of moving the patient without support for the neck and head is so

great that the splinting must be immediate must be of a type that can be applied with the patient flat on the ground and without the need of lifting his head and must be of materials that are easily and immediately available almost everywhere. These requirements are met by newspapers and small turkish towels. Either one can be used as an efficient emergency cervical splint. Before being applied, both are folded lengthwise in such a way that their folded width corresponds to the width between the angle of the



Fig. 25 The patient moved to the center of the bed. Note the attendant turning the patient's head.

patient's lower jaw and his clavicle. In either case one free end is pushed beneath the patient's neck, pulled through and wrapped snugly around and around until the paper or towel is entirely used. The wrapped material is then held in place by tying a string around it or by the use of adhesive tape. The splint must *not* overlap the lower jaw and must provide a shelf on which the lower jaw and occiput ride and by means of which flexion and extension of the head are prevented. With this splint in place the patient can now be rolled on his face with his forehead supported on a flatly folded

towel in such a way as to allow him to breathe and vomit freely. He should be left in the same place and in this position if transportation cannot be undertaken at once and then later can be safely rolled onto whatever stretcher or substitute therefor is used to transport him to the hospital.

Other than being transported face down and at full length with lateral steadying supports made of rolled blankets, large rocks, pillows, overcoats and so forth there is no occasion to provide emergency splinting for other levels of spinal-cord or spine injury.

Primary Hospital Splinting

First splinting of cervical injuries in the hospital should be combined with traction and should replace the folded newspaper or towel. The preferable method is with Crutchfield tongs (Fig. 26) (see page 235). These are



Fig. 26 Crutchfield type tongs in place.

miniature ice tongs in principle. The ice or head holding tips of the tongs are equipped with sharp points that are inserted into small holes made in the skull by a special drill point. The tong-points are prevented from slipping or from penetrating too deeply into the skull by appropriate locking

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devices. The tongs can be applied anywhere and the patient certainly need not be moved to an operating room for the purpose. The holes are drilled and the tips inserted just below and anterior to the parietal eminences in such a position that the pull they exert will be parallel to the normal long axis of the cervical spine and so located that they will be in as flat a surface as possible. After adjustment and locking, as much as 30 pounds can be hung from the tongs by means of a rope passed over a pulley. Through



Fig. 27 Flannel bandage halter in place.

adjustment of the height of the pulley the line of traction in relation to the line of the cervical spine can be parallel, in flexion or in extension.

At times Crutchfield tongs are not available and perhaps may not be procurable for some hours. This valuable time should not be wasted and until the tongs can be procured traction with a homemade flannel-bandage bridle should be substituted for the newspaper or towel splint (Fig. 27). This latter allows the use of a maximum amount of weight of only 5 pounds, however. To make the bridle, a flannel bandage $2\frac{1}{2}$ or 3 inches wide is folded lengthwise on itself so that it is of such a length as to make a loop around the patient's chin and still allow free ends extending beyond his skull for a matter of 4 to 6 inches. A similar but somewhat shorter length of flannel bandage is prepared in the same manner. The first bandage is then hooked around the patient's chin, with the free ends passing lateral to the

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ears. These free ends are then fastened to a wooden spreader the length of which slightly exceeds the approximate width of the patient's skull at its widest point. A hole is bored through the center of the spreader and threaded with one end of a rope passed over the pulley of a "Buck's extension" that has been fastened to the head of the bed (Fig. 28). The other



Fig. 28. Preferable method of attaching the Buck's extension apparatus to the head of the bed. Parts of a Balkan frame are used for the support of the pulleys.

end of this rope is tied in such a way as to support a five pound weight. With this apparatus in place the second loop of flannel bandage is passed beneath the patient's occiput, each end being brought out between the ears of the patient and the arms of the other loop folded over the top of the latter and held there with two safety pins at the diagonal corners. By tightening or loosening and upward and downward adjustment of this occipital

flannel loop and by raising and lowering the pulley of the "Buck's extension," the apparatus can be adjusted in such a way as to exert traction in any direction and to equalize the pull between the chin and occiput. No more than 5 pounds should be used with this apparatus, however because of the danger of producing pressure sores on the chin and occiput.

Primary hospital splinting for spine and spinal-cord injuries at other levels is adequately cared for by hyperextending the patient over a blanket roll that has been placed between bed boards on top of the spring and beneath a hair and a foam-rubber mattress. The roll should be placed at a point approximately opposite the kyphos. Its size will determine the amount of hyperextension (Fig. 9 page 71)

Splinting for Mobilization

Mobilization prior to weight-bearing and ambulation requires splinting that is designed to maintain the reduction and the position of the damaged

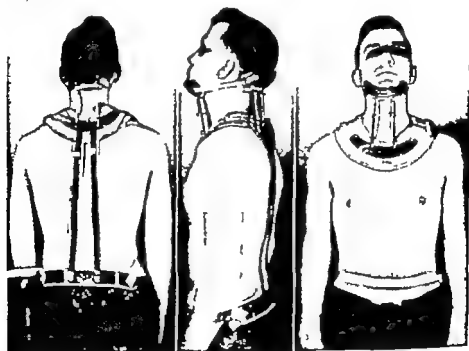


Fig. 29 A Zimmer type support modified by the attachment of pelvic band and back brace.

bone until the latter is solid, and yet afford the patient the benefit that comes from increased activity. In cervical-injury cases this requires the use of a Zimmer type support, modified to include a pelvic attachment (Fig. 29). The Zimmer-type support consists of two leather lined metal cups molded to fit the chin and occiput, respectively which are held on two threaded uprights that are adjustable for height. The cups are fastened to each other by leather straps at their sides. They are supported on anterior and pos-

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terior pieces of appropriately curved metal plates, which are attached to a ring of leather that rests on the clavicles, the shoulders and the back of the neck. This ring is divided into a front and a back half which are strapped together over the shoulders. Two strips of spring steel of appropriate width are attached by their upper ends to the posterior metal plate in such a way that they lie on either side of the vertical midline of the spinous processes. Their lower ends are fastened to a shaped padded piece of narrow spring steel that surrounds the pelvis just below the crest of the ilium and extends from just medial to the anterior superior spine on one side to a similar point on the other side. The two free ends of the pelvic girdle are buckled to each other by straps that are led through a suitably shaped abdominal pad. This combined splint will prevent the forward flexion of the head on the neck that is otherwise inevitable as long as the damaged bones are soft, the ligaments only newly healed and the posterior neck muscles too weak to support the weight of the head. The splint will not be efficient without the pelvic attachment. Properly applied it is as effective as supports of plastic or plaster of paris, is much more comfortable and does not produce pressure and bed sores or interfere with the respiratory exchange. It is infinitely more efficient than the so-called "Thomas collar."

Similar requirements for the rest of the spine are adequately met by the use of the ordinary Taylor back brace or Armstrong spring-steel type of brace.

Splinting for Ambulation

While the patients are being gradually mobilized within the limits imposed by the degree of healing of the bone injury steps should be taken to provide splints that will permit ambulation. This is particularly necessary for the quadriplegic, quadriparetic, paraplegic and paraparetic invalids. Rehabilitation in its final form depends on the patient's learning such mobilization as will guarantee him the ability to care for himself and, if there is no physical contraindication, the further ability to ambulate and to work.

Proper bracing or splinting for ambulation is the cornerstone on which the ambulation program is built, and ambulation plays a vital part in any comprehensive program designed to return the spinal paralytic to his right ful place in society. Of equal importance to relieve uncontrolled muscle spasm neurosurgical procedures designed to future efficient ambulation are. The proper treatment of spasm cannot be overemphasized. The paraplegic patient, deprived of most or all of his postural reflexes must develop through prolonged instruction and practice a new system of balance maintenance. This system is based on voluntary changes of posture designed to distribute body weight equally around the vertical axis of the skeletal system. The art of balance maintenance in sitting and standing is the first and basic phase of ambulation training and the entire success of subsequent ambulation instruction depends upon its acquisition. The spasm of an even moderately well developed mass reflex particularly in injuries of the thoracic

and cervical cord, renders this goal unattainable. Sudden, unpredictable stretch reflexes cause flexion or extension of the lower extremities and trunk, with precipitate loss of balance. The result is either a fall or a rescue by the instructor either of which offers a psychologic and mechanical hindrance to ambulation progress. Moreover decubitus ulcers, an ever-present threat in paraplegic patients, frequently result when braces are forced on a markedly spastic subject.

With these complications successfully treated, however, a contour tracing, supplemented by measurement of the circumferences of the extremities and the trunk, is made of the parts to be braced. When these measurements are made the patient must be lying face up on a flat surface. The prescribed brace is then given to the patient, and he is instructed in its application in bed. When this is learned together with other basic elements of self-care the patient is deemed ready to begin ambulation training.

A basic premise must be borne in mind concerning the factors involved in choosing the proper brace for a given patient. The function of the brace is not the support of body weight but the maintenance of normal postural relations through splinting action. In effect, the muscle groups responsible for maintaining the body in an erect, stable position are replaced by external mechanical supports, with maximum retention of normal joint function. Three determining factors may be mentioned. The first is the level of the injury which is usually the most important single criterion for determining the type of brace that the patient will need. When the optimal stage for brace fitting described above has been reached, there is usually a close correlation between the level of injury and the type of brace indicated. In most cases the type needed may be accurately determined in advance by careful muscle analysis to ascertain the existing deficit. Such tests should be done at regular intervals during the course of rehabilitation, not only for this purpose but as an index of improvement as well, particularly in partial lesions of the cord and cauda equina. Minor variations in the standard brace may be indicated thereby. Moreover the patient must be observed in all phases of ambulation training in order that a proper decision may be reached. Any changes are usually variations in a single category of brace rather than between categories—such as changes in the construction of a back brace.

The second factor is the severity of the injury whether it is a partial or a complete lesion of the cord or cauda equina. Selection of braces for complete lesions is relatively simple but partial lesions present a greater problem since the level of injury cannot be closely correlated with the type of brace indicated. For example, a patient with a partial cord lesion at the fifth cervical segment may require a double upright leg brace with a drop-foot spring on the right and a simple wire drop-foot brace on the left. These situations must again be met individually on the basis of thorough muscle analysis and clinical observation of the patient in ambulation; the appliance that most nearly replaces the existing deficit is then chosen.

The third consideration comprises malformations that may be present.

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Most of them will have been prevented or corrected surgically during the first three stages of rehabilitation but prolonged conservative therapy may be the treatment of choice. After relief of spasm such conditions as mild hip contractures and scoliosis, to mention two examples, may demand some special brace to permit corrective exercises and ambulation. These occasional complications seldom change the over-all requirements of a given patient but can be met by adjustments of a basic brace pattern.

Unless otherwise stated all levels referred to below are dermatome levels.

TYPES OF BRACES. *Drop-Foot or Short Leg Braces* (Fig. 30) The specifications for the drop-foot braces are as follows

WIRE DROP-FOOT BRACE (Fig. 30) The upright supports are made of 22 gauge steel wire and are attached to the shoe by a lightweight stirrup or

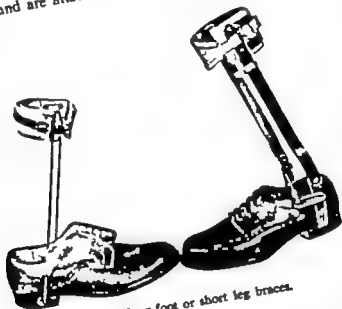


Fig. 30. Drop-foot or short leg braces.

piston through the heel. The calf band is 17 gauge spring steel with a $\frac{3}{16}$ inch sheet of sponge-rubber padding, covered with calfskin and held by single strap and buckle. The weight of the brace without a shoe is 1 pound 12 ounces; the total weight is 3 pounds 8 ounces.

DOUBLE UPRIGHT DROP-FOOT BRACE (Fig. 30) Steel uprights, $\frac{3}{8}$ by $\frac{3}{8}$ inch are used, with a calfskin band and strap similar to that of the wire drop-foot brace. A drop-foot spring is applied across a semihinged-type ankle joint with a stirrup shoe attachment. The weight of the brace without a shoe is 3 pounds, and the total weight is 6 pounds.

INDICATIONS. Analysis of the innervation of the muscles involved in control of the foot usually indicates that any loss of function below the knee results from an injury to the lowest lumbar or sacral-cord segments: the cauda equina, or all three. Paralyzes of this type are almost invariably flaccid, with considerable muscle atrophy due to damage of either the anterior horn cells in the fourth lumbar through the second sacral segments

or to transection of all or part of the filaments of the cauda equina. Spasm is rarely a problem. This injury is most frequently seen in patients with partial lesions of the sacral cord and cauda equina. In such involvement, the function of the brace is to substitute a compensatory force for the lost dorsiflexors of the foot and if necessary to stabilize the ankle. Paralysis of the

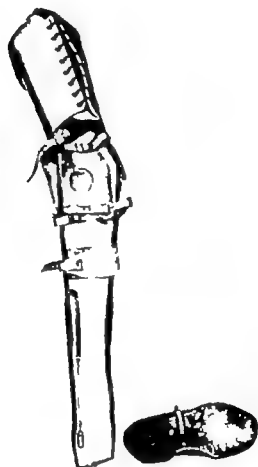


Fig. 31 Caliper type long leg brace with a detachable drop-foot spring attachment.

tibialis anterior, extensor digitorum longus and extensor hallucis longus muscles will result in a drop-foot with little change in ankle stability. Such patients, therefore, are adequately supported with the wire drop-foot brace, since no lateral stabilization of the ankle is required. In cases in which there is complete loss of all muscle activity below the knee, however, stabilization of the ankle is desirable, since such stability is normally chiefly dependent on the interaction of the muscle groups of the lateral and posterior aspects of the leg. This is best accomplished with the double upright drop-foot brace, which provides good bilateral support of the ankle joint and positive spring drop-foot correction.

Long Leg Braces (Figs. 31 and 32) The specifications for long leg braces are as follows

CALIPER TYPE LONG LEG BRACE WITH DETACHABLE DROP FOOT SPRING (Fig. 31) The uprights are made of $\frac{5}{8}$ by $\frac{3}{16}$ inch steel have a hinged knee joint with a drop-ring lock and calf and thigh bands of 17-gauge

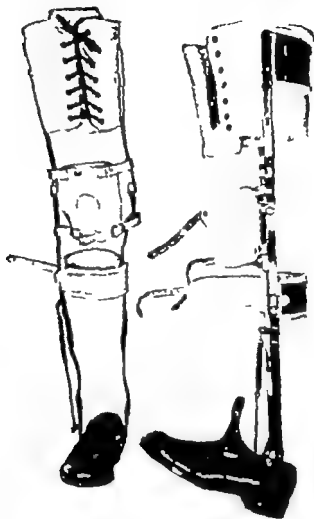


Fig. 32. Caliper type long leg brace with hinged ankle joint and stirrup attachment. Drop-foot spring and shoe are permanently attached.

spring steel, padded with $\frac{3}{16}$ inch sheet sponge rubber which is covered with calfskin. The ankle joint is of the caliper type with a detachable drop-foot spring. The drop-foot spring with a standard attachment above is fastened below to a steel instep bar measuring $\frac{3}{16}$ by $\frac{5}{8}$ inch by means of a detachable slotted plate that fits over a button forged on the end of the instep plate. The weight of each brace with a shoe is 5 pounds 8 ounces, and the total weight of both braces with shoes is 11 pounds.

LONG LEG BRACE WITH HINGED ANKLE JOINT AND STIRRUP ATTACHMENT (Fig. 32) The uprights are made of $\frac{3}{8}$ by $\frac{3}{16}$ -inch steel and have a hinged knee joint with drop-ring lock. The calf and thigh bands are 17-gauge spring steel padded with $\frac{3}{16}$ inch sheet sponge rubber covered with calf skin the drop-foot spring is permanently attached above and below on either side of a semihinged ankle joint. The weight of the brace without a shoe is 5 pounds 8 ounces and the total weight of both braces is 11 pounds.

INDICATIONS. The chief single indication for this type of brace is the loss of stability of the knees. This added factor marks the difference between the functions performed by this brace and those of the double upright drop-foot brace. The ability to maintain the legs in extension and consequently to maintain the body in erect posture is directly dependent on the integrity of the quadriceps femoris muscle. Should analysis and the clinical test of having the patient stand erect indicate that the activity of these muscles is lost or so weakened as to render extension of the legs inadequate, this function must be replaced by long leg braces. These deficits occur predominantly as the result of cord injuries below D9 segment but may be due to severe partial injuries or to complete transections of the cauda equina at or below the level of the third lumbar vertebra. Any remaining unparalyzed flexors of the leg are necessarily denied their normal action at the knee by virtue of the fact that the knee joint is locked in extension by the brace, and are not of primary importance in determining the type of brace used for injuries of this level. Their importance, as is true of any remaining function of the quadriceps femoris muscle lies in determining the type of gait the patient will use. This brace assists by enabling the patient with complete or partial loss of function from the ninth thoracic through the fifth sacral segment to stand erect and to be trained to use the gait best suited to his injury.

These two types of long leg brace are presented because of their relative merits and disadvantages. The first, with a detachable drop-foot spring, was devised in an effort to overcome the difficulties imposed by the caliper-stop brace while retaining the advantage of a detachable shoe. The caliper-stop brace moreover particularly with heavier patients and those actively engaged in ambulation, is often subject to recurrence of the drop-foot owing to posterior bending of the stops. This happens because all the ambulation gait—but particularly the swing-through—tend to cause forcible plantar flexion of the foot, extra stress therefore being exerted on the stops. Similar tension is exerted when the patient ascends stairs and other obstacles when, during the course of the ascent, the toe of the foot is caught temporarily under a protruding ledge. The detachable drop-foot spring type of long leg brace, on the other hand, has the drop-foot spring fastened permanently above but the lower attachments of the slotted plate to the protruding button on the shoe are made after the calipers are inserted. The positive tension exerted by such a properly adjusted drop-foot spring allows normal plantar flexion of the foot and has proved more effective than stops in correcting the drop foot. The spring should be parallel to its supporting lateral upright in order to reduce the tendency to eversion and abduction of the

foot, which may otherwise result if the mechanical advantage is too great. Furthermore the spring must be properly adjusted to prevent over-correction. The chief advantages derived are the convenience of application and removal, without the necessity of removing the shoes, and continued drop-foot correction. The advantage of the second type of brace with its hinged ankle joint and stirrup attachment, is the provision of a slight increase in lateral stability of the ankle with the same good drop-foot correction. The hinge may be constructed to allow plantar flexion of the foot. The obvious disadvantage of this brace is the greater difficulty of application and removal and the necessity for removing shoes and braces simultaneously.

In the use of either of these braces, three points of clinical importance should be emphasized. The hinged ankle joint should be placed 1 cm. above the tip of the lateral malleolus in line with the transverse axis of the talo-tibial joint. The knee joint should be placed opposite the midpoint of the medial and lateral condyles of the femur (the points of emergence of the transverse axis of the knee joint). If the knee joint is placed too high it will be noted on sitting that the thigh bands are displaced anteriorly and tend to cut into the soft tissues of the posterior surface of the thigh. The reverse is true when the joints are too low. There is a tendency though less marked, for similar pressure to be exerted on the leg with a displaced hinged ankle joint, and if the lower ends of the uprights are excessively high they may tear the patient's clothing when worn under street clothes. Care should be exercised in determining the height of the thigh bands. These should be placed on a slant with the lateral extremity 1 to 1½ inches higher than the medial end. Excessive height of the medial end may cause the formation of decubitus ulcers over the ischial tuberosities and undue pressure on the perineum and genitalia.

LONG LEG BRACES WITH A PELVIC BAND (Fig. 33) The specifications of the long leg braces with a pelvic band are as follows. The uprights are made of $\frac{3}{8}$ by $2\frac{3}{8}$ -inch steel with calf thigh and pelvic bands of 17-gauge spring steel, padded with $\frac{3}{16}$ inch sheet sponge rubber covered with calf skin. The ankle joint is of a caliper type and the knee joint is a drop-ring lock-hinge joint. The uprights are extended to a point midway between the great trochanter and the crest of the ilium with a free hinged joint allowing ante-flexion and retro-flexion at the hip incorporated opposite the great trochanter. The drop-foot spring is fixed above and is detachable at the shoe by means of a slotted plate that fits over the button of the instep bar. The pelvic band is secured by a single padded leather strap. The weight of the brace with shoes is 12 pounds 9 ounces.

INDICATIONS Analysis of the component parts of this brace indicates that its only additional feature comparing it with the long leg brace, is the pelvic band. This is attached by increasing the length of the lateral uprights of the long leg braces and fastening the band to the uprights with a freely moveable hip joint. No lock is attached. Its adoption for use by the paraplegic patient is based on certain limited but specific indications. Those injuries below D9 segment that are so complete as to deprive the patient of the functions dis-

cussed above under the indications for the long leg brace will in certain instances require the pelvic band extension in addition. The same foot and ankle support is needed, as well as the capacity to maintain the knee in extension in a standing position. The additional factors involved are the rotation deformities occasioned by the loss of part or all of the functions of

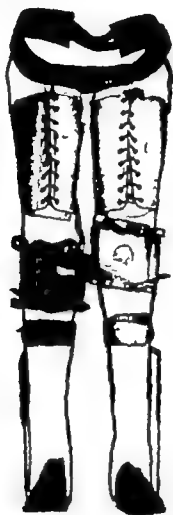


Fig. 33 Caliper type long leg brace with pelvic band. The hinge at the hip joint is freely moveable.

the internal and external rotators of the thigh. Paralysis of the external thigh rotators when combined with some remaining function of the internal rotators results in a medial rotation deformity of the lower extremities if long leg braces alone are used. This position renders it difficult for the patient to swing through smoothly and to regain balance properly during ambulation. Conversely unopposed external rotation of the feet interferes even more with these maneuvers. The specific function of the pelvic band is to correct either of these two forms of muscular imbalance by fixing the lower

extremities in the proper anteroposterior alignment of 5 degrees external rotation. As would be expected however no appreciable lateral stabilization of the hips can be obtained in addition since the chief point of such instability in high lesions is in the lumbar portion of the spine. Recognition of the need of a pelvic band may only be possible through a study of the patient's ability to maintain proper alignment of the lower extremities during ambulation.

It is essential that no lock be placed on the joint in the brace that is opposite the trochanter since the normal action of the hip joint must be preserved, regardless of the gait used. Moreover the hip joint of the brace must be placed at the proper level to obtain maximum function of that joint. When it is too high there is downward traction on the pelvic band, as well as undue pressure by the thigh band on the anterior aspect of the thigh when the patient is in the sitting position. If it is placed too low there is upward thrust on the pelvic band and pressure on the posterior aspect of the thigh. The pelvic band should fall midway between the crest of the ilium and the greater trochanter in order to avoid pressure over the bony prominences, with possible decubitus-ulcer formation. The comfort of the patient must also be considered in injuries that require this splint, because the top of the anesthetic area is usually below the point of contact between the body and the band.

LONG LEG BRACES WITH BACK BRACE ATTACHMENT (Figs. 34 and 35)

The specifications of these braces are as follows. The long leg braces and the lateral supports of the back brace are made of $\frac{5}{8}$ by $\frac{3}{16}$ inch steel, with calf and thigh bands of 17-gauge spring steel and a back band of 17-gauge spring steel or duraluminum, which is padded with $\frac{3}{16}$ -inch sponge rubber covered with calfskin. The ankle joints are of the caliper type, with detachable drop-foot springs. The knee joints have a drop-ring lock with a hinge joint, and the hip joints are of the freely moveable hinge type. Abdominal support is provided by a piece of 11-ounce duck fastened to the lateral steel elements with multiple canvas webstraps.

INDICATIONS The use of this type of brace is limited to the highest levels of cord injury—that is, those that are above D9 segment. The distinguishing features of the brace are lateral hip stabilization by functional replacement of the muscle groups controlling the actions of the pelvis and lumbar spine and functional unification of the thorax and pelvis as a substitute for the paralyzed trunk muscles. The degree of impairment of these muscle groups depends on the level and completeness of the injury. If analysis of the appropriate muscular activity indicates a deficiency of clinical importance artificial support must be used to replace the following vital functions: the maintenance of the erect posture of the trunk; the lateral stability of the pelvis and lumbar spine and the maintenance of the normal pelvic inclination. The first of these functions, or maintenance of the erect posture of the trunk, is performed by the iliocostalis lumborum, longissimus dorsi and quadratus lumborum muscles, which are all extensors of the spine and are predominantly innervated by the first four lumbar segments of the spinal

cord. They also act as lateral flexors by contraction of the homolateral with the simultaneous relaxation of the contralateral group. The second function—that of stabilization of the pelvis and lumbar spine—is provided by the intrinsic muscles surrounding the hip joints and the lumbosacral group

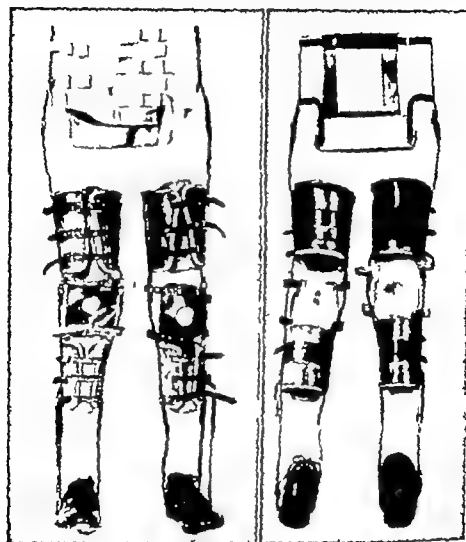


Fig. 34

Fig. 35

Figs. 34 and 35 Caliper type long leg brace with back support. Fig. 34 Front view
Fig. 35 Back view

of muscles. Although the former contribute appreciably to pelvic stability the latter appears clinically to be the more important. In addition to these extensor muscles, the flexors of the pelvis and lumbar spine are equally essential. They perform a dual function. They not only are important as flexors of the lumbar spine but also interact with the extensors of the pelvis and lumbar spine to maintain the normal angle of pelvic inclination. This angle formed between the true conjugate and the horizontal with the patient in the standing position is normally 50 to 60 degrees. Paralysis of

these two opposed groups occurs with transection at the sixth thoracic segment or above and results in lumbar lordosis with a marked increase in the angle of the pelvic inclination. This is increased by any hip-flexor contractures that may be present. Such lordosis is very disabling and virtually prohibits the attainment of balance and subsequent ambulation. The addition of the back brace is designed to correct this and to maintain extension of the trunk by articulation with the thoracic cage above and with the lateral leg supports below. The pelvic extension produced by the inferior transverse portion of the back attachment, together with the replacement of the abdominal musculature function by the abdominal support, tends to maintain the normal pelvic inclination. Side sway at the hips is eliminated by the lateral uprights, and thigh flexion and extension are retained through the freely moveable hinged hip joint (Figs. 34 and 35). The latter action is also indispensable in teaching the patient the proper swing-through in crutch ambulation. By being allowed free extension at the hip joint, the patient is enabled to balance momentarily with the thighs in slight extension after completing the swing-through while lifting his crutches forward to begin the next swing-through. The ability to get the hips forward after completion of one swing-through in preparation for the next is essential for efficient swing-through ambulation, regardless of the type of brace employed.

It is in connection with the use of this brace that the necessity for proper preambulation therapy becomes most evident. Any spasm of the muscles acting on the thigh, pelvis and trunk must be relieved otherwise, the patient will be subject to sudden "jacking" forward or backward as the result of a mass reflex initiated by the stretching of these muscles in the act of swinging. The patient must also have completed the basic maneuvers of balance, since it is particularly important in lesions of this level that stability and a feeling of confidence be acquired. Should all medical therapy of a well developed mass reflex fail and the patient refuse surgical correction this type of brace cannot be employed consequently the most efficient ambulation cannot be attained. The alternative is the long leg brace with a pelvic band but with locks on the hip joints and a wider abdominal support. This combination allows the patient to substitute flexion and extension in the lumbar spine for normal hip motion because of the splinting effect provided by his spastic muscles. This is not an efficient method of ambulating, and this treatment is never recommended as the procedure of choice but only as a last resort under these particular circumstances.

The height of the back brace has been the subject of much discussion and experiment, but the optimum has proved to be 4 cm. below the inferior angle of the scapulas (Figs. 36 and 37). This is sufficiently high to allow good articulation with the lower portion of the thoracic cage and yet is low enough to permit free action of the scapulas and other elements of the pectoral girdle. Higher back braces are superfluous, since the thoracic cage is structurally well supported by ligaments and the pectoral girdle. A positive objection to braces extending above this point is the interference offered

to the proper use of crutches, which become entangled with the back-brace extension. The height also has a direct bearing on the total weight of the splint, which at best exceeds that of any other type. Any attempts to reduce the weight by substituting duraluminum for the uprights and transverse struts of the back brace have resulted in failure because of the great stress that the appliance is called on to bear. Some weight, however may be

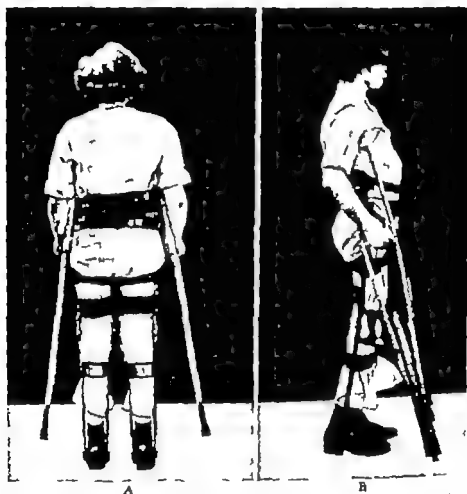


Fig. 36. Patient wearing caliper type long leg brace with a back support. Standing
A back view B side view

spared by the use of sheet duraluminum instead of 17-gauge steel in the main body of the back support, but further use of this material is foredoomed to failure.

The caliper with a detachable drop-foot spring was developed primarily for patients with back braces, since the stirrup-type leg brace with shoes and back brace attached requires the assistance of one or two persons in the application and removal of the unwieldy device. With calipers and a detachable drop-foot spring the patient can wear shoes continually insert the calipers into the shoes and then proceed to lace the brace from below

upward. Unless a caliper lock is employed in addition to stabilize the ankle joint, some stability may be sacrificed, however. This is justifiable since it means the difference between a brace that is practicable for use and one that is not.

PREFABRICATED ANKLE JOINT FOR LONG LEG BRACES A prefabricated ankle joint for the long leg braces that combines the stability of the caliper stop-lock and the flexibility of the detachable drop-foot spring has recently come on the market. It is sold under the trade name of the Pope Foundation

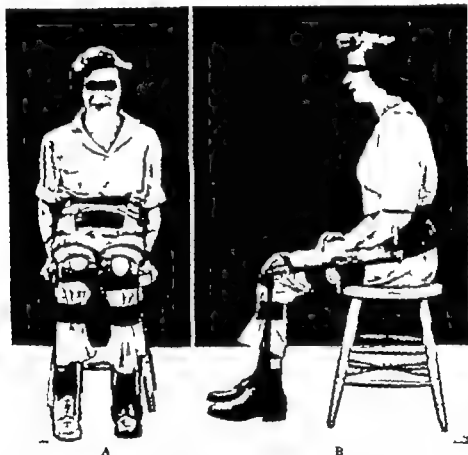


Fig. 37 Patient wearing caliper type long leg brace with a back support. Seated A front view B side view

197 South West Ave. Kankakee, Illinois, and is designed to be incorporated into the lower end of the lateral upright of the long leg braces between that point and the shoe attachment. It is so built as to provide solid lateral support for the ankle to be detachable from the base plate in the heel of the shoe and yet to provide the desirable mobile spring-type of drop-foot correction by virtue of an incorporated coil-spring opposite the ankle joint. The tension of this spring is adjustable to suit the individual's need. So far after fairly extensive use this seems to offer the best solution of the problem posed by the need for support for drop-feet in patients that can wear long leg braces. The added expense is not prohibitive.

Miscellaneous (Fig. 38) In patients whose injuries are above the first thoracic segment, certain special appliances made for the arms may prove useful, depending on the distribution and amount of arm muscle paralysis that is present. Among these are the triceps brace used to maintain the arms in extension; a leather mitt with straps to hold the paralyzed fingers in flexion around the crutch-grip (not shown) and a leather wristlet to maintain wrist stability. Others are the knee strap that is used when necessary to prevent

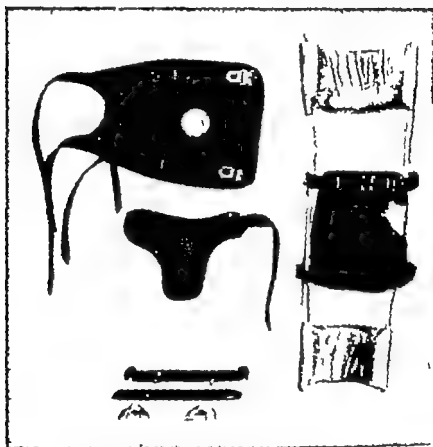


Fig. 38 Miscellaneous braces. Upper left, knee strap; middle left, "T" strap; lower left, detachable spreader bar; right, "sleeper" brace.

genu varus or genu valgus, and the "T" strap that performs a similar function at the ankle joint. A cock-up splint is made of a wire frame with transverse canvas web supports. A sleeper or a temporary brace for use on the legs and the cock-up splints are used in the earlier stages of mobilization before the patient becomes ambulatory. They aid in preventing flexion contractures of the wrist, ankle and knee. The detachable spreader bar is occasionally employed in teaching the swing through to patients in class 4, but its further use is discouraged. It is attached by either end to both medial uprights at the ankle level by means of bolts and wing nuts and promotes simultaneous forward movement of the legs. The Taylor back brace (not shown) is used for the correction of spinal deformities in the occasional

which severe kyphosis develops in high thoracic and cervical

It be noted that the indications for splinting for ambulation are based
ly on the assumption of a complete transecting lesion at the level
ed. In incomplete or partial injuries of the cord below the cervical

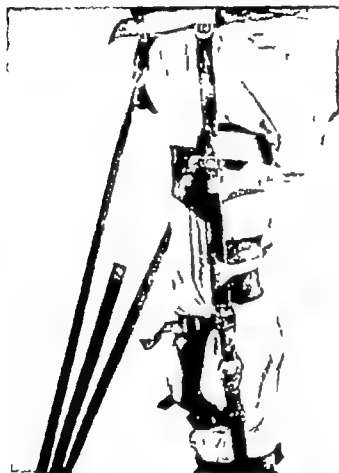


Fig. 39

Fig. 39 and 40. Construction of knee and hip joints in caliper type long leg braces
either a pelvic band or a back support. Note the mechanical method of locking
knee joint and the freely moveable nonlockable hip joint. Fig. 39 The patient is
it and the knee joint is locked.

and of the cauda equina, it is impossible to state categorically in
ance exactly what type of appliance will be indicated, not only because
e wide variance in the initial pattern of muscular dysfunction but also
use there may be gradual improvement in muscular power over a period
onths following the trauma. Muscle analysis is indispensable as the
1 step in deciding on the splint requirements when the patient is ready
racing. With this information at hand and with the specific function
rmed by each type of appliance borne in mind, a rational approach
be made to each individual problem. In cervical cord injuries the

extent and distribution of the paralysis in the arm muscles will determine the appliances necessary to attain the greatest amount of useful motion.

Brace Construction

The knee lock used in the long leg braces is recommended after testing several more complicated types (Figs. 39 and 40). It is of simple, rugged construction and requires only a minimum of machining and repair. This



Fig. 40.

Fig. 40 The patient is seated and the knee joint is unlocked.

gravity lock is easily manipulated and foolproof during use. More elaborate locks are more expensive, are liable to damage by twisting or bending and may lead to unexpected falls.

The width of calf and thigh bands is of practical importance. As a general rule, best results are obtained from narrow bands. Circulatory embarrassment may well result in older patients if calf and thigh bands are too wide or laced too tightly. This precaution is particularly applicable in patients with any changes in or other impairment of the peripheral circulation.

Steel is the material of choice although its weight is somewhat greater than that of duraluminum. Its durability and capacity to withstand torsion and stress particularly those of the higher braces and in heavy patients, make it irreplaceable by any of the light alloys at present available. Furthermore, observations on duraluminum braces indicate that joints made of this metal deteriorate rapidly and that the frequent twisting of the uprights

necessitates constant adjustment, which is rarely if ever completely successful.

Gaits

It is essential, in any discussion of braces to mention the type of gait that may reasonably be employed with a given appliance. Patients requiring only drop-foot braces will have the most nearly normal gait. Their ultimate requirements may be limited to one or two canes, and they will vary from normal only in that they will tend to proceed on a rather wide base with steppage gait. Initially it may be necessary to train such patients with crutches instead of canes, but the use of crutches should be discouraged as rapidly as possible—the most obvious objections to them being that they call attention to the patient's handicap and promote a feeling of dependency.

The type of gait to be employed by patients with long leg braces will depend on the degree of function remaining in the thigh flexors. Should these muscles be adequate to advance the legs even though they are not strong enough to support the patient's body weight, either the four-point or the more rapid two-point crutch gait is feasible. In either case, the patient should also be taught the swing-through, which is the most rapid of all gaits and which may be required in situations in which speed is desirable, as in crossing streets at traffic lights. This type of gait can be taught to any properly prepared patient with a complete or partial lesion below the first thoracic segment and is mandatory for those with loss of thigh flexors and with higher paralyses. The pelvic band group will not usually have enough function remaining to allow them to use the two-point and four-point gaits and consequently must be taught the swing-through with the preliminary temporary swing-to gait.

For patients requiring back braces, the only practicable gait for distance walking is the swing-through, which should be taught to all such patients except those with lesions above the second thoracic segment that are so severe as to cause gross impairment of arm function. As in the pelvic-band group the swing-to is taught as a preliminary to learning the swing-through. The swing-to is retained only for maneuvering in close quarters where the lack of space renders the swing-through impossible. Since their hip joints must be moveable for efficient ambulation, it is incumbent on these patients to exercise more skill and balance than is demanded of those with lower injuries.

No patient should be permitted to attempt either the shuffle or a swing to gait until he has thoroughly mastered the art of balancing with first one and then both crutches off the floor unsupported either by an instructor or by artificial means. The poise and sense of confidence gained thereby is a basic requirement in the later progress through the shuffle and swing-to and in the acquisition of an efficient swing-through gait. Concurrently and later all patients must go through a period of intensive mat work and other calisthenics designed to strengthen the muscles of the pectoral girdle and arms

to a point that will permit them to meet without fatigue the stress of weight bearing that is demanded for ambulation. This is particularly important in the group with the higher cord injuries.

Lesions high enough to involve the nerve supply to the muscles of the upper extremity usually force the patient to walk with a swing-to or shuffle gait.

Discipline

Patients with severe spinal-cord or cauda-equina injuries often present difficult disciplinary problems. They and those closely related to or professionally associated with them may have extremely difficult adjustments to make. These are psychological first of all, and then social, financial and operational. Successful adjustment is only possible to all concerned if the ultimate goal is kept always in view. This goal is to provide the patient with the greatest possible amount of rehabilitation. Psychologic rehabilitation is possible in its fullest sense only when physical rehabilitation is as near perfection as possible. This applies not only to the adjustment required of the patient but to that required of his immediate family, his attendant or nurse, his doctor and his friends as well.

The foundation of physical rehabilitation is the ability to care for one's self. It is during this early period of training that the problem of discipline may appear. Any instance of lack of self-discipline, which may be manifested as refusal to eat, the use of foul language, liquor drinking, throwing things or physical violence, is fundamentally an emotional outburst that is quite analogous to temper tantrums in a child. Attempts to correct it by sympathy, the application of reason, acquiescence or physical combativeness only make bad matters worse. The only effective way of dealing with this problem at this time is to isolate the patient physically, deprive him of his audience and withdraw all services until the emotional storm is over. There should be no hesitation about this. The wife, attendant or nurse should give him clear warning at the first of such outbreaks that when the next occurs she will drop what she is doing, whether it is feeding him, cleaning or bathing him, giving him an enema, changing his bed or anything else and neither return nor resume her activities until an apology has been offered and accepted. There should be no recriminations, fixing of blame, nagging or punishment as an aftermath, however. A prerequisite to successful discipline—and especially to a maximum rehabilitation—is a complete knowledge by the patient, and especially by his immediate family of his condition and all that it implies. There is no greater detriment to the adjustment and rehabilitation of a paralyzed patient than a husband, wife or parent who refuses to face the facts and continues to live in a dream world that includes the impossible. A corollary to this important statement is that before he makes a prognosis the doctor must know the facts and be able to explain them fully, factually, unemotionally and understandably to all concerned.

Further disciplines are those of self-control, determination, courage, hard

physical labor unwillingness to accept undeserved sympathy and a burning desire to earn community respect by retaining and maintaining self respect. Means toward these ends are the learning of self-care the acquisition of maximum mobility ambulation if possible, the acquisition of a gainful occupation and proper adjustment to uncorrectable physical deficiencies. As Franklin D. Roosevelt is said to have remarked "the only permanent deficiencies are in the head"

Miscellaneous Problems

Spasm in Conjunction with Retained Voluntary Motion

Perhaps the most important (and still unsolved) problem affecting paraplegics and quadriplegics is the treatment and correction of any disabling spasm that is associated with such a degree of voluntary motion that if the latter were divorced from the spasm it could be put to good practical use in all the various activities of rehabilitation and especially in those of self care and ambulation. This spasm differs in no way from the spasm seen in the body area below the level of an anatomically transected cord, which has been described under the general term "mass reflex." It is predominantly adductor-flexor in type but may be chiefly extensor or a combination of the three. I have never seen any abduction phase in this condition. The spasm does not occur in connection with uncomplicated cauda-equina injuries. These spastic motions are set off by any minimal noxious stimulus. This causes spreading muscular contractions that are followed by a residual hypertonus in the contracted positions. It is best and most typically elicited by applying the noxious stimulus to the foot, ankle or lower leg but in severe cases may be caused by the lightest of tactile stimuli on any point of the body or extremities that is neurologically below the spinal segments that have been injured. The cord injury in the type of case under discussion may be compressive or noncompressive, is a partial injury and may be at any level from the fifth cervical to the fifth sacral segment. Not enough cases have been observed to determine as yet what the commonest level is if any.

Although invisible the spasm affects the large paravertebral muscles and those that form part of the posterior abdominal wall equally as severely as it affects the surface muscle groups. If severe it is painful and its prolonged presence will cause tendon contractures, joint deformities that will be permanent, and gradual diminution of the effectiveness of the associated retained voluntary motion. Electrical stimulation of such spinal cords in the spinal segments above the injured area will produce active contractions of the muscles of the legs and lower trunk, thus indicating intactness of some of the tracts leading from above the damage to the final common pathways below it. It is certain that the spasm can be abolished (in company with the voluntary motion) by spinal anesthesia. It is unaffected by chemical interruption of the sympathetic-nervous-system pathways. It is not affected by destruction of the pelvic parasympathetic nerves. It can be completely or partially abolished in the legs by procaine injection of the sciatic, the obturator and the femoral nerves in the thigh for as long as the anesthetic

and analgesic effects remain. During the anesthetic period voluntary motor control is also lost but returns with disappearance of the anesthetic effect. In the mild cases abolition of spasm may be permanent even after the paralysis has disappeared and especially after several injections. Injections of the sciatic and other lower-extremity nerves and the resultant relief of spasm in the legs do not affect the spasms in the trunk or arm muscles, however.

Mild spasm in the trunk and legs may be relieved by a decompressive laminectomy over the site of the cord injury. This relief does not always occur, however. In one patient the pain of severe spasms in the arms has been done away with by partial rootlet rhizotomy, but the arm spasms themselves have not been affected otherwise. In another patient cervical rootlet rhizotomy permanently abolished the spasms without any loss of voluntary motion. Injection of the sacral roots with procaine for a spastic detrusor and of the internal pudic nerves for a spastic external urethral sphincter (see pages 104 and 105) has had no effect on any spasm other than that of the bladder and sphincter. The spasm, whether mild or severe, can also be temporarily diminished or even abolished by exhausting the spastic muscles through overexercise. Thus *maximum physiotherapy* is effective for a time and will remain so as long as the muscles are in a state of exhaustion. With rest, however, the spasm returns in severe cases. In mild cases it may be diminished or even done away with. Injection of the brachial plexus with procaine has been ineffectual in severe arm spasm in that the temporary relief was minimal, did not affect either the trunk or leg muscles and was as severe as ever after the anesthetic effect had worn off.

The cause of these spasms—not only in the patients with a partial cord injury but also in those with a transected cord—has not as yet been demonstrated. It is known, however, that resection and lysis of the upper and lower cut ends of a transected cord at the point of transection gives relief of spasm only during the period of spinal shock. After this has passed off the spasm returns as before.

The treatment is discouraging and baffling. The first essential, however, is to know definitely whether or not the cord injury is a transecting or a partial one. This makes exploration and electrical stimulation of the cord mandatory as a first step before any direct treatment can be intelligently conceived or put into effect (see pages 228 and 231). If there is compression of the injured segments, an adequate decompressive laminectomy must also be provided (see pages 231 and 234). If these procedures demonstrate that the cord is transected nothing is to be gained by postponing an anterior dorsolumbar rhizotomy as a method of dealing with spasm of the trunk and legs. If the cord injury is a partial one but the associated residual voluntary motion below the level of injury is apparently so little as to be ineffective even in the absence of spasm, an anterior dorsolumbar rhizotomy must eventually be performed. Since any delay in doing so will incur the risk of permanent deformities of tendons or joints, only a minimal postponement before its performance should be countenanced.

If on the other hand the partial cord injury is so minimal that a high degree of residual voluntary motor control is present in the legs and trunk a permanently destructive operation such as a rhizotomy is not permissible and recourse must be had to peripheral neurotomies, tenotomies, myelotomies and the like supplemented by procaine injections and physiotherapy to the point of muscle exhaustion. These measures should especially include active mat exercises and persistent active ambulation.

No intelligent advice concerning the treatment and relief of trunk and leg spasm in the group of patients that fall between the two extremes of anatomical transection and minimal partial injury of the cord or in the group of patients that have disabling spasms of their arms regardless of the severity of the cord injury can be given in the light of our present knowledge. Everything except permanently destructive procedures should be repeatedly tried; the patient should be encouraged to make the most of what possibilities he has and the profession should be stimulated to study and restudy this problem with a view to its eventual solution.

Flexion Contraction at the Hips and Associated Lordosis

This complication is definitely the result of neglect. It may be that the patient failed to follow the doctor's advice. It may be that the doctor failed to order proper physiotherapy soon enough. It may be that the physiotherapist was incompetent, or it may be—and this is the usual cause—that everybody concerned just didn't think it worth while to make any effort at more than day-to-day therapy. This *laissez faire* attitude springs from the conviction that "since nothing can be done to relieve the paralysis and the patient is a permanent invalid anyway the sooner he dies the better." Nothing could be more unjustified and more unworthy of all concerned than this point of view; nevertheless, neglect of this or of one of the other opportunities leads solely and directly to what may be a very disabling and unnecessary deformity. This type of lordosis is especially likely to develop in the presence of the spasm that may be associated with a transected cord.

Cause. It comes about by virtue of the contractures that develop in the iliopsoas, the tensor fascia lata and the sartorius. The first two are the most important. When these muscles are permanently shortened, the pelvis is tilted forward in an effort to permit apparent extension of the hip. Varying degrees of lumbar lordosis develop. A patient with this deformity is very much more difficult to splint in preparation for ambulation than if it were not present, and many such patients cannot ambulate at all even if splinted. Since, for practical purposes the deformity is present only with a cord transection above D9 segment, the splint must of necessity include a thoracopelvic cage in addition to the two calipers (see page 145). The fitting of this cage and the finding of a material that is stiff enough to withstand the stresses exerted on it is time-consuming and difficult (Fig. 41). Because of lack of understanding and the difficulties of dealing with this problem, efforts to correct or compensate for the deformity are usually

abandoned and the patient is improperly classed as one for whom ambulation and its accompanying mobility are not possible.

The *diagnosis* is made by the finding of a significant lordosis of the lumbar spine (Fig. 42) and is verified by the following maneuver: The patient is put flat on his back on a hard surface. Both legs are flexed at the hip as far as possible. This will flatten the lumbar spine and place the pelvis in its proper plane. One leg is then maintained in full flexion and the other is extended at the hip to the point at which the extension can-

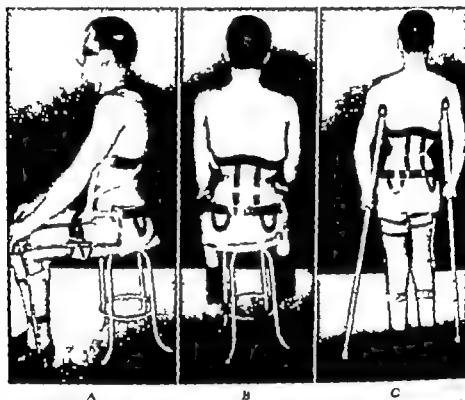


Fig. 41 Caliper type long leg braces with back support modified to compensate for a severe lumbar lordosis. *A* patient seated, side and *B* back view *C* patient standing, back view

not be carried further without tilting of the pelvis. The resultant amount of elevation of the heel above the flat surface will indicate the degree of fixed hip-flexion. The examination is repeated for the other leg with the position of the legs reversed.

Treatment Treatment must do two things. The fascia-lata and deep thigh fascia must be divided horizontally so thoroughly that there are no longer any palpable fascial bands under tension and the iliopsoas must be separated from or divided at its attachment to the lesser trochanter. In severe contractures this latter division should be complete, but in the less severe ones or where there is still a residual of voluntary control of the muscle one can, as recommended by Peterson¹⁴ content himself with dividing

only the tendon and the medial half of the muscle. Even with these extensive resections, continuous and active physiotherapy must be continued for a long time in the hope that the scar will not retract enough to cause a repetition of the deformity. This should include stretching the scar by hanging the patient's legs over the side of the bed. He should be lying

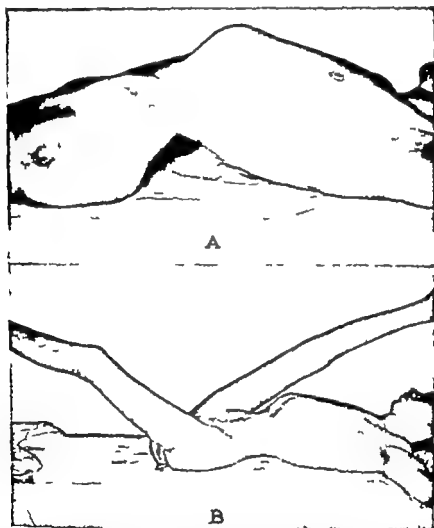


Fig. 42. *A* compensatory lumbar lordosis produced by uncorrected contraction of the flexor muscles of the hip. *B* partial correction of the lordosis by full flexion of the right hip joint. Note the extreme contraction of the hip flexors on the left as evidenced by the unsupported position of the left leg.

transversely across the bed on his back, with his buttocks on the edge. This maneuver should be carried out at least every night and every morning for significant periods of time.

The prognosis, even with this active therapy is not too favorable in neglected cases—and almost all cases that show this condition have been neglected. The cut tendons and muscles reunite with scar tissue, the scar

tissue contracts and unless the basic spasm has been eliminated by permanent neurosurgical procedures it will reproduce the original deformity in a surprisingly short time and in spite of every active effort to prevent it.

Other Contractures

Other contractures in the knees, ankles and toes of paraplegics and quadriplegics and in the shoulders, elbows, wrists and fingers in quadriplegics are also evidence of neglect. In the lower extremity mild cases of spasm that do not warrant an anterior dorsolumbar rhizotomy or in which the destructive operation is contraindicated because of residual significant voluntary motion can be dealt with by tenotomies and myotomies, nerve crushing or nerve-sectioning with or without immediate suture procaine or alcohol paraspinal injections or surgical division of the appropriate spinal roots. If necessary and only if it does not endanger the vascular supply to the limb by deprivation of its splinting effect, capsulotomy may also be done. It must always be borne in mind, however, that significant fixed, old contractures of the tendons about a joint and of the capsule may be associated also with compensatory shortening of the large limb vessels that pass close to the joint on its flexor surface. Too rapid release of these stabilizing and splinting muscles that, by their presence, have prevented dangerous overstretching of these vessels may cause thrombosis or even rupture with consequent loss of the limb below. To at least help in obviating this danger preliminary extension of the joint with dependent weights or with the use of spring-steel, elastic or turn-buckle extension splints must be given an adequate trial prior to this surgery. This is particularly true of the upper extremity where vascular occlusion would be a tragedy of the first order. It is extremely doubtful whether it is ever justifiable to permanently stabilize any joint by arthrodesis. The reasons are obvious as regards the upper extremity. In the lower extremity however specious arguments may be advanced in its favor—notably the diminution in the amount of necessary splinting. The surgeon should not allow himself to be persuaded to consent on this or any other ground, however, in an arthrodesis of either the knee or ankle. The knee if properly arthrodesed, keeps the leg in extension (Fig. 43). Since it cannot be moved out of the way its protrusion into the pathway of others is a source of great annoyance with some danger to the patient. It is also a major detriment to proper ambulation and self-care, just as an arthrodesed ankle is. The objections to this superficially attractive procedure are not theoretical they are practical, and are the result of sorrowful experience.

In the severe or moderately severe contractures with spasm, too much must not be expected of the peripheral operations proposed for their correction. Reinnervation earlier than expected, the effect of neural overlap, the reunion of cut tendons and muscles with contraction of the uniting scar and the resumption of spasm with its deleterious effect on the joints involved, are predictable and frequent if not usual end results. Anything better comes as an unexpected dividend. It is preferable in these cases,

unless there is a significant amount of voluntary muscular control to proceed at once to doing an anterior dorsolumbar rhizotomy thus doing away with the basic causative factor. The residual contractures can then be dealt with by peripheral operations, with some reasonable hope of permanent relief.



Fig. 43. Arthrodesis with fusion of the right knee joint. Note the resultant inconvenience.

Periarticular Calcification

The cause of this condition is not apparent. It does not appear to lie in tearing of the periosteum or muscles. It may be associated with hematomas in the muscles, and it is probably a function of the abnormal calcium metabolism associated with clinical and subclinical genitourinary tract infection. I have never seen it except in those neglected cases in which genitourinary-tract infection was all too easy to demonstrate and was of long standing. Motion in the joint does not affect its occurrence. The calcified areas slowly enlarge to a certain size that is unpredictable in advance. No therapy that influences their progress is known. The mass may or may not be so intimately associated with the joint that it involves the joint ligaments, or it may be placed at a distance laterally or posteriorly. I have never seen or heard of this type of calcification in the paralyzed upper extremities. Excision for cause—such as reactivating flexion in a blocked joint—may be tried. It is an unsatisfactory bloody and usually inefficient procedure however in that there tends to be a prompt recur

Physiotherapy does no harm, nor do mat exercises, ambulation or self-care activities. The most effective treatment devised to date is to leave the calcified areas alone, treat the genitourinary tract effectively and mobilize the patient as early and as much as possible. The diagnosis is made by x ray examination.

Phlebitis Thrombosis and Emboli

From present available figures it appears that phlebitis venous thrombosis and resulting pulmonary emboli occur in between 5 and 8 per cent of all types and levels of injuries to the spinal cord and cauda equina. Phlebitis and thrombosis occur in both femoral and spinal veins, and pulmonary emboli may be loosed from either source. In addition, advancing necrosis of the cord tissue that has been caused by major interference with its venous circulation may follow femoral as well as spinal venous thrombosis. When the diagnosis is made the standard treatment of ligation of the phlebotic thrombosed vein at the classical sites should be resorted to. Experience is lacking in the efficiency and use of anticoagulants such as heparin and Dicumarol. In these cases of spinal-cord and cauda equina injury it is obvious that prolonged immobilization of the lower extremities plays no part in the production of venous thrombi or emboli.

Respiratory-Tract Infection

The importance of preventing respiratory-tract infection in patients with upper thoracic or particularly with cervical cord injuries cannot be overestimated. The problem is a mechanical one and depends for its significance on the inability of the patient to cough up the secretions formed in the respiratory tree. This applies not only to the lungs but also to the trachea, the larynx and the pharynx. The cervical patients especially deprived as they may be through paralysis of the use of the intercostal and accessory respiratory muscles, and being obliged to depend on the diaphragm alone for the forcible expulsion of bronchial tracheal and laryngeal exudates as well as those that drip down the upper respiratory tract from the pharynx, nose and nasal sinuses, will drown in their own secretions unless prompt measures are taken to assist in their removal. In the milder cases stimulation of the cough reflex, liquefaction of the exudate by steam inhalations, postural drainage and suction of the pharynx under direct vision may be effective. Such drugs as atropine and of course the sulfonamides and antibiotics should be used as necessary and indicated. The administration of oxygen as well as all measures that are required for the maintenance of cardiac and circulatory competence are essential. Tight strapping with a circular adhesive plaster strapping, as described elsewhere in this section (see page 67) greatly increases the effectiveness of coughing and should not be neglected. All other measures described above (see page 67) to increase oxygenation must also be practiced. Despite all this, however, it may be still necessary especially in the patients with a cervical cord transection, to empty the reachable portion of the lungs by bronchoscopy. There

should be no hesitation in employing this procedure as often as necessary daily and even twice daily evacuation by this means being usually essential. This is a procedure that must be carried out only by a skilled and practiced bronchoscopist, however because the risk inherent in the required positioning of the patient with the strain on the damaged and possibly dislocated cervical vertebrae and cord can only be kept within reasonable limits through the greatest dexterity and gentleness. As a prophylaxis which in the last analysis is better than any treatment this group of patients should be carefully quarantined from all visitors with any respiratory tract infection however mild.

Hyperhydrosis

Many but not all patients with spinal-cord injuries will suffer from being constantly bathed in perspiration. The sweating area may be but is not necessarily limited to the parts of the body that are above the paralyzed section. Sweating is also an accompaniment of the syndrome of visceral distention sweating and rise in blood pressure (see page 117). The cause of this extremely annoying disability is unknown but, *a priori* appears to be associated in some way with a disturbance of the sympathetic nervous system. The type and level of cord injury appears to have no bearing on the occurrence of hyperhydrosis. No certain method of control is known. It may stop or decrease, without evident cause after a laminectomy the relief of spasm, or the institution of control of the bladder and bowel. Medicinal applications to the skin have no influence. Constant bathing of the skin in perspiration does not macerate the skin, nor does it predispose to pressure or bed sores unless the perspiration is mixed with urine. There is a possibility that it may be associated with loss of body temperature control, as I have noticed that certain patients who have sweated profusely earlier in their disease, later cease to perspire and take on the temperature of their surroundings. They appear to have lost their ability to either cool themselves in a hot atmosphere or warm themselves in a cold one. Thus inability to sweat may be a problem in hot weather because of this disarrangement of the mechanism that normally leads to reduction of body heat by evaporation of perspiration. Air-conditioning of the room or ward has been the only solution in such cases. Hyperhydrosis and anhydrosis do not occur in pure cauda-equina injuries.

Gynecomastia Testicular Atrophy Abnormality of Liver Function and Abnormalities of Renal Function

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Abnormality of or interference with the liver function is said to occur in male paraplegics. Verification and, even more, understanding of this observation await further study.

The rate of secretion of urine from the kidney varies within wide limits in certain paraplegic and quadriplegic patients (see page 97). This is a highly complicated problem that is closely allied to circulatory efficiency, bladder function, fluid intake, temperature control, sweating and infection. The surface of this problem has not even been scratched, and virtually nothing is known about the matter at this time.

Flaccid Scoliosis

Quadriplegic children who have been rendered so by a cervical cord injury suffer from a scoliosis of the thoracolumbar spine that can only be described as flaccid. In this condition the spine assumes S curves in any direction whenever the attempt is made to have such patients sit erect (Fig. 44). The deformity does not become fixed, as far as my experience has shown, and it can be straightened out and the spine returned to its normal configuration by traction on the head with the patient upright—provided that the bone injury is healed—or by placing him flat on his back. This lack of support to the spine is obviously primarily traceable to the total flaccid paralysis of all the paraspinal muscles, which is characteristic of quadriplegia. Secondarily, however, the elasticity and instability of the ligamentous and joint structures of the vertebral column during childhood must be a factor as well. In support of this theoretical concept is the fact that I have never seen this kind of scoliosis in any adult with a comparable injury.

Its presence is a major detriment to the self-care activities that are necessary if any degree of rehabilitation is to be granted these patients. They cannot sit erect, hence cannot learn to balance and hence are greatly handicapped in making the most of what is left of the musculature of their hands and arms. The associated abdominal compression caused by the slumping also interferes with bladder rehabilitation.

I do not know of any satisfactory treatment. Plastic jackets fitted from a plaster mold made with the patient held erect by head-traction have proven the most satisfactory in maintaining the normal spine configuration when the patient is sitting, but these have obvious disadvantages. Among them are the necessity of continued replacement to match the child's growth, their heat in summer, the difficulty of getting a "skin fit," and the inevitable development of pressure sores under the weight bearing points. Surgical fusion appears to be impractical. Plaster of Paris jackets are worse and more destructive than plastic ones, and steel-back braces of the Taylor type are ineffective. Much more ingenuity should be used in the solution of this problem than has been apparent to date.

Painful Hands

Quadriplegics will occasionally complain of constant burning and boring pain in their paralyzed hands. There will be nothing characteristic about

the signs and especially there need not be any local evidence of vasospasm or vasodilatation. Injection of the stellate or the upper three thoracic sympathetic ganglia with procaine will confirm the cause as minor causalgia (see page 205)



Fig. 44 Flaccid scoliosis, patient sitting and lying.

Repeated injections with procaine may give permanent relief. If they do not and especially if the injection site becomes indurated or if the period of relief grows continuously shorter after each injection more active therapy must be instituted. Before doing this however it is well to be

sure that the causalgia does not spring from an irritated injured cervical root (see pages 177 and 179) Stereoscopic oblique roentgenograms should be taken to show the cervical intervertebral foramina in cross section therefore and a cervical myelogram should be done Any evidence of significant occlusion of the appropriate intervertebral foramen or foramina or of protrusion or extrusion of a cervical disc with compression radiculitis, should lead to appropriate decompressive surgery as a preliminary to further and more radical interference with the sympathetic ganglionated chain.

If there is no evidence that compression or irritation of the cervical roots is the cause of the pain or causalgia or both, then no time should be wasted before injecting the first three thoracic sympathetic ganglia with procaine if this has not already been done or doing a surgical resection of these ganglia and their intervening chain. The prognosis gets increasingly poor with the postponement of active therapy

Flipper Hands

Elderly people who have been rendered quadriplegic by accident, particularly if they have been undernourished for any significant length of



Fig. 45 Flipper hand. Upper hand, flipper; lower hand, normal hand for comparison.

time prior to their injury as well as similar young adults whose treatment has been neglected, will frequently develop a deformity of their hands that can best be described by the term "flipper hands" (Fig. 45) The fingers are fixed in extension with little or no movement in the joints, the normal hollow of the palm disappears and the hand and fingers look as if they had been flattened out by pressure This appearance seems to be traceable to the atrophy and loss of contour of the thenar and hypothenar eminences

and of the normally rounded palmar prominence that overlies the distal end of the metacarpal bones at the base of the four fingers. In the aged, there will be x-ray evidence of arthritic changes. Once established, this condition is highly resistant to treatment. The usual physiotherapeutic measures of passive and, if possible assisted active motion, manipulation of the joints, massage in various forms stretching of contracted tendons and the like should all be pressed vigorously. The patients must be made to ingest large amounts of all the vitamins in addition to a full, properly balanced diet. In addition it appears to be helpful to provide stimulus to the large vessels in the upper thorax and supraclavicular regions by local diathermy. It may be that this increases the circulation of the extremity. This complication is very infrequent, and it has not been observed enough to warrant recommendation of the use of ACTH cortisone or thoracic sympathetic block. The prognosis is probably bad in any event.

Therapeutic Amputation of Paralyzed Legs

It has been suggested that it is more practical to amputate the paralyzed legs of all paraplegics than to make the effort to mobilize the patients with their legs in place. Such a suggestion is repeated here only because it is so bizarre and so out of accord with the facts and with the modern approach to rehabilitation that it merits notice for the single purpose of utter condemnation. Any person suggesting such a solution to the problem of paraplegia only demonstrates his total ignorance of the problem as a whole, as well as a complete unfamiliarity with the surgical advances of the past ten years. Therapeutic amputation of the legs of paraplegics when proposed or performed as a method of dealing with their paralysis is unforgivable.

Leg amputations in paraplegics are justifiable only when the amputation is part of the necessary treatment of the original injury or when osteomyelitis has destroyed the acetabulum head neck and upper end of the shaft of the femur to such an extent that the leg can no longer bear weight. Whether the leg is or is not paralyzed has no bearing on the indications for amputation.

Paraplegics who have lost one or both legs, however need not despair of mobilization on that account. It has now been demonstrated by actual experience that prostheses can be substituted and used in place of the absent extremities in the types of self-care general mobilization and crutch walking that are otherwise suitable for a paraplegic.

I have one patient in whom a prosthesis of the bucket type has been successfully substituted for a non-weight-bearing leg (Fig. 46). The patient had a transection of the lumbar cord and his leg was amputated through the hip joint because of osteomyelitic destruction of the joint and upper end of the femur. I have also seen at the Kessler Institute at West Orange New Jersey three men patients with cord transections in the thoracic region and double mid thigh amputations with paraplegic stumps. One was fully trained and ambulant with the skills of class 6 including climbing stairs

both up and down. He wore two prostheses. Another also wore two prostheses, to which he had just been fitted. He was able to stand erect on them with the aid of crutches. The third was like the second, except that he was less steady in his balance.

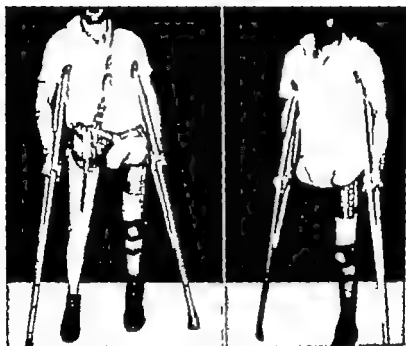


Fig. 46 The use of a prosthesis to replace a leg amputated through the hip joint in a paraplegic. Patient with and without the prosthesis.

Amyloid Disease

Amyloid disease will develop in paraplegics or quadriplegics from the same causes that bring it about in chronic infectious diseases. The only difference is that it occurs sooner in these patients with additional chronic spinal paralysis. Whenever it is present it may be regarded as evidence of neglect of the patient in the earlier stages of the post-spinal-injury period. Here too sepsis that has been out of control and stayed so for a year or more is the basis for the amyloid disease. Its lethal effect is enhanced by any hypoproteinemias or avitaminosis that may have been permitted to develop or by any anemia that may have been allowed to continue untreated. The diagnosis is made by the demonstration of the characteristic chemical changes in the urine and blood serum and a strongly positive Congo red test.

Treatment of this type of amyloidosis is ineffective. The best therapy is prevention together with adequate well conceived and properly executed treatment of the fundamental sepsis. The prognosis is that of the condition that caused the amyloidosis in that if the prognosis of the former is bad the latter is also but if that of the former is good the amyloidosis may cease to progress and in some instances may even regress with corresponding benefit to the patient.

Combined Cranlocerebral and Spinal Cord Injuries

This combination occurs sufficiently frequently to make it necessary for the surgeon to always bear it in mind as a possibility. It is most likely to take the form of a cranlocerebral and cervical spine or cord injury although any level of cord damage may be associated with the head injury. Once the combination is thought of the diagnosis is apparent because the signs and symptoms of the respective injuries differ in no way from those seen when either lesion is present alone. As far as treatment is concerned the most important consideration is to remember that when there is a cervical injury present it is important to provide fixation of the spine in extension before proceeding to treat the cranlocerebral injury. Other than that, all diagnostic procedures and therapeutic methods should be carried out as directed in the appropriate sections under the respective headings.

Functional or Anatomical Union of the Severed Spinal Cord

It should be clearly understood that incontrovertible evidence exists that demonstrates conclusively that once the human spinal cord has been divided at any level it cannot be made to unite and will not unite except by scar tissue. The passage of neural impulses from levels above such an anatomical division to segments below the injured area, with resultant return of either somatic sensation or voluntary motion is permanently out of the question. No matter how much time is allowed to pass, the barrier will remain complete. Apparent anatomical severance deduced from such data as the time degree and extent of the paralysis or sensory loss can not be verified by clinical signs alone however. Complete physiological interruption of impulses has been indisputably shown to be present in the early stages of cord injuries in patients who later have recovered virtually normal sensation and voluntary motor control in what was originally a totally paralyzed area. *Per contra* patients who have retained a certain degree of sensation and voluntary motor control for a few days immediately after a cord injury only to permanently lose both these functions with the passage of time are not uncommon. In the first group the functional loss is traceable to local edema without destruction of the cord tissue and hence is followed by recovery. In the second, a partial injury deteriorates to a destructive complete transverse myelitis because of the death of previously living tissue from ischemic necrosis, venous thrombosis, swelling and pressure necrosis. It is axiomatic that a certain differential diagnosis between a permanent and a temporary transecting myelitis can only be made by exposure of the cord for the purpose of electrical stimulation above the damaged area (see pages 228 and 231) or by the passage of time. Spasm, whether flexor or extensor or both, is not diagnostic of the pathology of the cord injury. Once it has been established and has been permanent for a matter of six to eight weeks in a demonstrated and proven anatomically transected cord however it cannot be expected to disappear or alter for the better unless the patient goes into spinal

shock. The reverse is also true—that is, that once the patient is out of spinal shock and has been free of spasm for six to eight weeks, and provided that an anatomical transection of the cord has been proven to be present by appropriate electrical stimulation at a laminectomy the spasm can be counted on not to appear later in the course of the disease. On the one hand nothing is to be gained by postponing surgical measures—such as an anterior dorsolumbar rhizotomy—for its relief when spasm is present as described above nor on the other hand, should the need for such operative relief be anticipated in the future in the absence of spasm after the initial two months have passed, *always providing* spinal shock is not present during that period.

The Treatment of Injuries to the Cranial Nerves

These patients ordinarily must be hospitalized for treatment.

General Considerations

Cranial nerve injuries are almost exclusively associated with craniocerebral injuries or occur as the result of damage at operation.

Except for the fifth nerve it is impossible to recognize, for therapeutic purposes, damage to the various nuclei of origin of the cranial nerves. When such damage does occur it is always a part of the greater and more widespread cerebral damage that is of itself paramount in the determination of treatment. Certain cranial nerves have long, unsupported courses along the floor of the skull or are so situated as to be in close proximity to usual fracture lines. Among the former are the oculomotor nerves and among the latter the olfactory, the trigeminal, the auditory, the facial and the optic nerves. These geographical characteristics have much to do with the determination of the frequency with which the individual nerves are injured.

The Olfactory Nerve

Injury to this nerve occurs as part of a craniocerebral injury. It is *untreatable* except as a part of the more extensive injury.

The Optic Nerve

Injury to this nerve occurs as part of a craniocerebral injury or as the result of unintentional or deliberate injury in the course of an orbital or intracranial operation. Care should be taken to warn the patient or his responsible relatives of such injury at the earliest possible moment. The injury is *untreatable*.

The Third, Fourth and Sixth or Oculomotor Nerves

Damage to these nerves is commonly associated with craniocerebral injuries involving the base of the brain or the floor of the skull or occurs as the result of intracranial operations involving the anterior and middle fossae. Isolated paralysis of one sixth nerve also occurs, according to Cogan¹³ after spinal anesthesia. Treatment of the nerve injury itself is ineffective but much can often be done by appropriate ophthalmologic

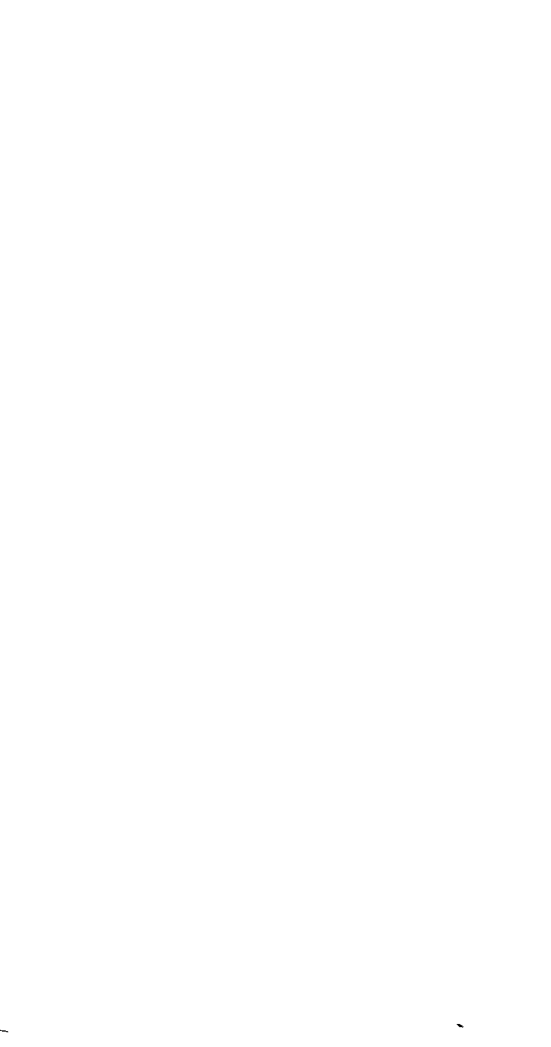
surgery. The patients should be referred promptly to an ophthalmologist for this purpose. It is fortunate that the prognosis for recovery in these cases is always better than it appears to be. As a result, operations on the external ocular muscles designed to correct deformities resulting from their paralysis should be delayed until all chance of reinnervation has vanished following recovery of the involved nerve from its original injury. The isolated sixth-nerve paralysis associated with spinal anesthesia (see page 215) needs no treatment, as it recovers in some weeks of its own accord.

The Trigeminal Nerve

Injury to the trigeminal or fifth cranial nerve is commonly associated with injuries to the face and forehead and with operations on the jaws, the Gasserian ganglion and its root. There may also be an injury to the central part of the nerve in the upper part of the spinal cord and in the medulla. The *symptoms and signs* include paresthesias, hyperesthesia, hypesthesia and anesthesia in the sensory distribution and motor paralysis of the motor portion. *Treatment.* Injuries to the tract in the spinal cord or medulla are untreatable. Injuries to the motor root are also untreatable. Injuries to the sensory root are untreatable as such, but the dry and insensitive cornea that accompanies injury to the fibers that eventually make up the first division must be treated. The eyelids should be sutured and the eye covered until such time as ulceration of the cornea is no longer to be feared. After that (usually within a month or six weeks) the lids can be separated again, but the patient should wear goggles or some similar protection, especially when the eye is exposed to dust or wind. An ophthalmologist should have charge of the eye during this period. The paresthesias of the face that not infrequently follow injury to the sensory root are virtually untreatable, as is anesthesia in the same area from the same cause.

Injuries to the ganglion produce virtually the same problems as the root injuries, except that the complications arising out of the dry anesthetic eye are more stubborn and more difficult to deal with. The treatment is the same as that for the symptoms caused by injury to the sensory root.

Injuries to the intracranial portions of the three sensory branches are untreatable except for the eye complications, which should be handled as described above. Injuries to the extracranial branches cannot be repaired. Persistent pain in the area supplied by the second and third branches may require intracranial surgical section of the sensory root for relief, no other treatment being possible. The supraorbital and infraorbital nerves, if injured externally, should be dealt with locally if necessary because of pain. In the case of the supraorbital nerve, any local scar should be excised and the proximal end of the divided nerve isolated, crushed and tied with silk. If possible the supraorbital artery should not be disturbed, because of the danger of producing paresthesias, attacks of sharp pain and local flushing apparently traceable to overactivity of the autonomic fibers that travel with or in the wall of the vessel. Treatment of these latter symptoms is notoriously unsuccessful. Pain following injury to the infraorbital nerve can some-



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times be relieved, after exposure of the nerve at the infraorbital foramen by crushing and ligation if the trunk is not too frayed or damaged.

The Facial Nerve

This is damaged as part of a cranio-cerebral injury as the result of ear operations, at the point of damage or operation in the region of the parotid, as a complication of the temporal extradural approach to the ganglion and root of the fifth nerve, or after exposure of the side of the face to cold or a persistent draft. It may also be injured in the posterior fossa by accident, or otherwise in the course of an operation for Ménière's syndrome or for the removal of a cerebellopontine-angle tumor. The treatment of injuries of the intracranial portion is out of the question. The best that can be done is to correct as well as possible the resulting facial paralysis. These efforts are largely limited to various plastic and sling operations on the face itself.^{14, 17} They should be left to the specialist in plastic surgery. Injuries to the nerve in the fallopian canal can, in certain instances, be repaired by grafting within the canal^{18, 19, 20, 21} or by grafting the cut end of the distal part of the nerve to the cut end of the central part of the surgically divided hypoglossal or spinal accessory nerves in the neck.^{22, 23} If the latter anastomoses are successful, motion will be restored to the paralyzed facial muscles but will be accompanied by annoying and frequently embarrassing involuntary motions of the tongue, shoulder, eye or head. The restored motor function will be limited to useful motion and will never be completely normal. The plastic and sling procedures can also be used in these instances. The same general methods apply to injury of the main trunk in the parotid region. Attempts at suture are only rarely successful. Injury in the course of an operative extradural exposure of the ganglion and root of the fifth nerve in the temporal fossa result from hemorrhage in the hiatus fallopii. The facial nerve is not divided in these instances but is compressed by a blood clot in the canal. The lost function usually returns in six to eight weeks after the clot has organized. Support for the facial muscles during the time that they are paralyzed should not be neglected, however. In all cases, and until return of function takes place or permanent support is provided for them, overstretching of the paralyzed muscles can be prevented to a worth-while degree by frequent massage of the face in an upward direction from the corner of the mouth toward the outer canthus and temporal region by the patient himself. Electrical stimulation at regular intervals is also helpful and maintains the contractility of the paralyzed muscles. Bell's palsy or a functional interruption of the facial nerve from exposure will practically always recover without active therapy directed toward the nerve itself.²¹ Massage and so forth, of the muscles during the time they are paralyzed is essential, however.

The Nervus Intermedius

This part of the facial-nerve complex is undoubtedly injured at times in the course of severe cranio-cerebral injuries and is certainly injured unin-

tionally every so often in the course of the surgical division of the vestibular nerve in the treatment of Ménière's syndrome. Its division in the posterior fossa causes no demonstrable signs or symptoms, and it is no amenable to treatment.

Intentional division in the posterior fossa as a method of treating intractable atypical facial pain is said to have been successful.²⁴ Such a procedure should be undertaken—at present, at least—only as a last resort and on an experimental basis. Too few patients have been subjected to such therapy to justify its use on any other grounds.

Injury to this nerve at any other point in its course causes no recognizable signs or symptoms and is untreatable.

Hemifacial Spasm

Henry Woltman and H. L. Williams²⁵ of the Mayo Clinic have recently postulated a neuritis of the facial nerve as the cause and have successfully treated on the basis of that postulation certain cases of hemifacial spasm. While the work is not final it is so promising and the alternative therapeutic measures so ineffective that it is worthwhile calling attention to it.

Patients with hemifacial spasm suffer from irregularly recurring, nearly constant tic-like spasms of the muscles of one side of the face. One group of these patients, at least, develop these signs and symptoms following recovery from Bell's palsy. Treatment has hitherto been ineffective. Woltman and Williams, acting on the theory that there had been a traumatic intracanalicular neuritis with irritation and compression of the trunk of the facial nerve in the bony canal medially from the stylomastoid foramen, unroofed the canal in the mastoid and demonstrated a compressing fibrous sheath surrounding the nerve trunk. When the sheath was split in its long axis, a reddened swollen nerve bulged through the slit. No further local therapy was given except that great care was taken not to remove the sheath or the nerve from the canal and thus endanger the blood supply to the nerve. The postauricular wound through which the approach was made was closed as usual. All their patients were afforded prompt, complete and permanent relief during several months follow-up. Any such surgery should be done by a specialist in otology.

The Auditory Nerve

The Cochlear Portion

This is usually damaged as part of a craniocerebral injury. In the course of an operation for Ménière's disease or during the removal of a cerebello-pontine-angle tumor. Treatment is impossible.

The Vestibular Portion

Injury to the vestibular nerve must be considered from the points of view of both injury to the trunk and injury to the vestibule itself. Injury to the trunk takes place by the same mechanisms that cause injury to the cochlear portion of the auditory nerve, and, in addition because of pur

poseful surgical division in the treatment of Ménière's syndrome. No *treatment* is possible.

Injury to the vestibular apparatus may occur as a part of damage to the internal ear. As a result, the patient will have the typical signs and symptoms of loss or alteration of vestibular function. The most bothersome will be dizziness, which is only persistent with partial destruction of the apparatus. Total destruction of the semicircular canals will cause temporary dizziness that is self limited and that requires no active therapy other than avoidance of invalidism and immobilization. Partial destruction of the labyrinth may require *treatment* if the symptoms persist for more than six to eight months after the patient is again active. The disabling vertigo, unsteadiness on turning corners and lack of stability in relation to the patient's position in space are analogous to a nontraumatic Ménière's syndrome. It should be treated in the same way. Conservative measures should be employed first. These must include splinting of a flapping drum, maintenance of an open eustachian tube, the limitation of salt ingestion and the administration of small doses of the barbiturates. If the symptoms do not subside in six months under this regimen, surgical division of one vestibular nerve in the posterior fossa may be carried out.

The Glossopharyngeal Nerve

Injury to this nerve occurs so infrequently and causes such a paucity of signs and symptoms as to be virtually unrecognizable. *Treatment* is impossible.

The Vagus Nerve

This is usually damaged as part of a craniocerebral injury in the region of the medulla and pons near the point of origin of this and the eleventh nerve in the course of an operation for removal of a posterior fossa tumor or in the neck in the course of an operation on the thyroid gland or other operative procedures in that general area. *Treatment* of the nerve injury itself is impossible but prompt attention must be paid to any respiratory obstruction resulting from the cadaveric position of the vocal cords. Inflation pneumonia must be carefully guarded against. Intubation or tracheotomy must be performed promptly when indicated. The laryngologist should be consulted at once and he should be given full charge of the treatment for this condition as well as for the loss of or alteration in the voice that is produced by this lesion.

The Spinal Accessory Nerve

This is usually damaged in the course of extensive craniocerebral injuries, the removal of a posterior fossa tumor during any operation from the region of its point of origin from the brain stem and spinal cord to its emergence through the jugular foramen in the skull or during a dissection of the neck. Intracranial injuries are *untreatable*. Division of the nerve in the neck, when recognized, should be dealt with by immediate end-to-end

suture. Support for the arm and shoulder and splinting of the neck must be provided in addition. Drooping of the shoulder and displacement of the scapula resulting from paralysis and atrophy of the trapezius and sternomastoid in old unrecognized cases should be referred to the orthopedic specialist for treatment.

The Hypoglossal Nerve

This is usually damaged as a part of severe craniocerebral injuries, in the course of an operation for removal of a tumor of the posterior fossa or in the course of surgical operations on the neck. Intracranial injuries cannot be treated. Extracranial injuries, if recognized, should be dealt with if possible by prompt end-to-end anastomosis of the divided nerve. Oral hygiene must be scrupulous and watch must be kept lest the patient choke himself or inhale foreign matter because of the paralysis of the tongue.

The Treatment of Injuries to the Spinal Roots

All these patients must be hospitalized for treatment (See also section on *Injury to the Spinal Cord and Cauda Equina*)

General Considerations

The spinal roots, other than the cauda equina are those parts of the nervous system that start as rootlets and that then coalesce and extend laterally as anterior and posterior roots from the posterolateral and anterolateral aspects of the spinal cord. The latter pierce the spinal arachnoid and dura, acquiring thereby a sheath of both membranes. The roots end at or in the intervertebral foramina just distal to the posterior root ganglion at the point where the anterior and posterior portions become fused to form the peripheral nerves. The first cervical root has no posterior or sensory portion in a high percentage of cases, is hidden behind the uppermost attachment of the dentate ligament and may have an anastomosing branch from one of the smaller branches of the spinal accessory nerve. Sympathetic and parasympathetic fibers travel with the anterior portion of the spinal or somatic root. Symptoms and signs of damage appear peripherally in the form of motor paralysis of the flaccid or final-common pathway type with more or less exact distribution in accordance with myomeres or in the form of pain and sensory abnormality including paresthesias, hyperesthesia, and anesthesia corresponding more or less exactly to the limits of the appropriate dermatomes. Involvement of the sympathetic fibers of any one root will be unrecognizable as such because of the wide intraneural connections that exist in this system. Involvement of the spinal parasympathetic fibers is considered under *Injuries to the Spinal Cord and Cauda Equina* (see page 56)

Acute Nontransecting Spinal Radiculitis

This is commonly associated with fractures and dislocations of the vertebral column or with stab and gunshot wounds of the spinal canal or paravertebral area, or as the result of trauma to the neck in the newborn at delivery. It may or may not be associated with injury to the spinal cord. The diagnosis is presumed and usually made from the history, the x ray findings, the presence of pain or paralysis in the appropriate dermatome or myomere and the complaint of constant burning or sharp shooting pains in the general peripheral area corresponding roughly to the region supplied

by the root or roots in question *Pathology* The root may be compressed, distorted or partly divided. The compression or distortion may be from bone fragments, narrowing or distortion of the intervertebral foramen or dislocation without fracture of adjacent vertebrae. Irritation or physiologic or partial anatomic division, with or without tearing and possibly associated with local hemorrhage will result. *Treatment* Direct treatment is usually limited to the operative removal of any aberrant compressing agent, such as a fragment of bone or a foreign body. The approach varies with the individual problem and is conditioned by the associated injuries. If the damage has been caused by a stab or gunshot wound the tract should be debrided as part of the procedure. Chemotherapy and antibiotics should be used both locally and parenterally. Suture of partially divided or torn roots is only rarely possible or successful. Such direct operative therapy should not be undertaken unless the preoperative diagnosis is reasonably accurate.

Any nonoperative indirect type of therapy—which is the usual method—is best carried out by the use of any effective treatment of the associated fracture or dislocation of the spinal column. This will nearly always call for traction, hyperextension and fixation of the spine (see page 63).

Acute Transection of a Spinal Root

This follows the same types of injury that are described under *Acute Nontransecting Spinal Radiculitis*. The diagnosis is presumed and usually made just as in the previous section, except that actual transection can only be verified by surgical exposure and electrical stimulation of the root in question. Transection will be evident through failure of stimulation of that part of the root that is central to the injury to produce appropriate peripheral motor and sensory responses. *Pathology* There is a complete transection of the rootlets or of the root at any point in its course. Such divided roots do not regenerate if sutured. Neuromas do not form on the cut end of the central segment. *Treatment* There is no effective treatment leading to recovery of function of any totally divided spinal root. However pain caused by compression by scar of the cut end of the proximal segment can be treated at times with a varying degree of success by surgical neurolysis.

In all instances in which the diagnosis of total anatomic division of the root has not been verified by visualization and electrical stimulation, the root should be presumed to be capable of sufficient inherent ability to repair itself and the structures to which it is distributed peripherally should be kept in as nearly normal a functional state as possible until all chance of such repair with its consequent resumption of function, is out of the question. This may not occur until after a year has elapsed following the injury although evidence of returning function will usually manifest itself earlier. The normal tonicity, contractility and length of any paralyzed muscles as well as the normal mobility of any joints that may be involved, must be maintained during this period. Physiotherapy, splinting in the position of optimum use and passive and active assisted motion must be religiously carried out. These procedures can be supplemented, if necessary by periph-

eral electrical stimulation of the muscles and by measures designed to promote the circulation of the extremity. With the first return of voluntary motion active use must be carefully restricted to that which is well within the limits of muscular exhaustion. Splinting should be continued until danger of overstretching or distortion of the weak muscles by their non-paralyzed antagonists or because of the dead weight of any distal part of the limb is obviated.

After it is apparent that no repair of the injured root is going to take place or after surgical exposure has verified the presence of a root transection and it is evident that the functional loss is permanent, appropriate orthopedic procedures such as tendon and muscle transplants, tenodeses, reinnervation by nerve transplant and arthrodeses may all be considered. The needs of each case will be highly individualized and therapy must be provided accordingly.

Chronic Nontransecting Spinal Radiculitis

This is commonly initiated by an injury to the vertebral column that was considered minor at the time of its occurrence. Repeated similar injuries, each of which produces symptoms that last longer and that are increasingly severe and each of which causes increasing pathology produce symptoms that tend to become progressively more disabling over a period of time until the patient finally demands adequate diagnosis and effective therapy. The usual causes are a rupture of a cervical or lumbar disc, with protrusion and then extrusion of the nucleus pulposus with invasion of the intervertebral canal, or increase in the amount and calcium content of the tissues that surround the joint between the adjoining facets, with resultant narrowing of the intervertebral foramen. This causes an increasing compression of the appropriate root.

Ruptured Intervertebral Disc

Lumbar

The diagnosis is suggested by the history and physical findings. It is confirmed by the demonstration of an appropriate defect in a column of radiopaque material that has been previously introduced into the lumbar subarachnoid space. *Pathology* There is rupture of the posterior portion of the annulus, and possibly of the posterior spinal ligament as well, with at first protrusion and later extrusion of the nucleus pulposus in such a direction as to irritate, deform or compress one particular root in its intervertebral canal or deform or compress and possibly irritate any part or all of the cauda equina at the appropriate level. There is frequently an associated unstable relationship of the lumbosacral or interlumbar articular facets and some degree of so-called "hypertrophic arthritis," neither of which plays any part in the etiology, the pathology or the production of the symptoms. Frequently there is also a narrowing of an intervertebral disc space. This is confirmatory but not of itself diagnostic of an extruded nucleus or ruptured

disc. Motility between adjacent vertebrae demonstrated at operation and thought to be either abnormal in amount or extent is not a sign of either a ruptured disc or a protruded or extruded nucleus. Multiple ruptures of discs with multiple protrusions or extrusions with appropriate defects in the diagnostic study of the radiopaque subarachnoid column occur with greater frequency than would be expected. Distortion or compression of the appropriate roots will be found at operation. *Signs and Symptoms* The symptoms are those of any acute low-back condition superimposed on a background of chronic low back pain with irritation and spasm of the paravertebral muscles, together with pain that spreads down the distribution of some part of one sciatic nerve. In certain cases (midline extrusions) the symptoms may involve an area corresponding to the peripheral distribution of more than one root and may even spread over parts of both legs. The signs that go with spasm of the paravertebral muscles, acute and chronic arthritis of the intervertebral joints and irritation or interruption of one or more roots are suggestive but are not diagnostic unless confirmed by a properly done myelogram. *Treatment* In the earlier stages, and usually before the diagnosis is more than suspected these patients should have the benefit of conservative nonoperative therapy. This should be carried out under the direction of an orthopedic specialist. It should consist of graduated exercises combined with rest and possibly fixation of the spine by appropriate splinting in the hope that the protruded nucleus will recede, the rent in the annulus close and a subsequent rupture with recurrence of symptoms be prevented by the development of greater stability and increased local range of motion, all made possible by an increase in the strength and effectiveness of the great muscles that support the spine and pelvis.

Once the diagnosis is established, or once it has been decided (in the doubtful cases) to make the diagnosis by exploratory laminectomy however and a ruptured disc with a protruded or extruded nucleus that deforms a root or the cauda has been demonstrated, the opening through the annulus and the posterior ligament should be enlarged sufficiently to permit the removal of all disc tissue as well as the opposing surfaces of the vertebral bodies. Care should be taken not to perforate the anterior wall of the disc, however, as this will, in all probability, damage one of the iliac vessels and may lead to an otherwise unnecessary leg amputation. The diagnosis and even the demonstration at operation of a ruptured lumbar intervertebral disc does not of itself justify or indicate fusion of the spine. The decision concerning the need for this procedure should be made as if the disc had not been involved and the fusion was needed or not needed only because of other appropriate indications.

The postoperative care is as important to the eventual outcome as the operation itself. Appropriate exercises that will stretch without irritating the shortened irritable back and leg muscles as well as the scar in the back must be faithfully practiced. In addition graduated increasingly severe exercise of a general nature should be prescribed.

Cervical

The *diagnosis* is suggested by the history and physical findings and is confirmed by x-ray demonstration of a significant narrowing of the appropriate intervertebral foramen through the use of oblique films, and by a constant appropriate deformity in the outline of a column of radiopaque dye introduced into the cervical subarachnoid space. Narrowing of the disc space may also be observed by x ray. *Pathology* There is a rupture of the annulus and, rarely, of the ligament overlying one of the lower four cervical discs, with either compression of the anterior aspect of the cord through the overlying dura or narrowing of the appropriate intervertebral foramen. Protrusions and extrusions of nuclei from ruptured cervical discs are prone to calcify and form solid irremovable tumors anterior and lateral to the cervical cord and its roots. The so-called "hypertrophic arthritis" is usually present and either may or may not contribute by its presence to the signs and symptoms. *Signs and symptoms* The signs and symptoms are those of irritation or interruption of any appropriate cervical root, or irritation or local destruction by compression of a part of the structures in the anterior cervical cord. Pain, sensory abnormalities, muscular weakness, atrophy and so forth in the neck, shoulder, arm and hand are characteristic of this disease if a root is involved. If the cord is compressed there will be signs of involvement of the long tracts. *Treatment* In the patients with milder signs and symptoms it is worth while to devote a reasonable period of time to conservative therapy. This is carried out not with any idea of changing the shape or size of the protrusion or extrusion, but rather in the hope that, through lessening the irritation of the soft tissues that surround the facets and the intervertebral foramen, any swelling that is present will subside and the tissues will become more elastic and the paraspinal muscles less irritable. As a result, the irritation and compression of the root may be reduced to a point where the symptoms and signs will largely disappear. The methods used in this conservative therapy are those that provide support for the weight of the head on the shoulders rather than by way of the cervical spine, after any necessary relief of muscular spasm has been accomplished by traction. The former include the Thomas collar and fixation of the cervical spine by other more effective splinting. The Zimmer type of cervical brace, to which has been added a posterior member that fixes the splint to the patient's pelvis, is the preferred splint (see Fig. 29, page 136). The latter is accomplished by skeletal traction or by bed rest with hyperextension with or without traction. If, after a reasonable time, it is apparent that conservative means have been ineffective, direct operative therapy must be resorted to. The involved area is exposed by a minimal hemilaminectomy, the dura opened and the cord rotated sufficiently to expose the compressed root after division of the necessary number of lateral attachments of the dentate ligament. Unless the protrusion or extrusion is easily exposed by extradural dissection and then as easily mobilized, no attempt should be made to remove it. This is particularly true if it is calcified. The risk of accompany

ing irreversible damage to the cord or roots is too great otherwise. If such a tumor is in the midline and irremovable two or three of the lateral attachments of the dentate ligaments should be cut on both sides to allow for greater mobility of the cord, and the dura posterior to the cord should be left open. If the tumor is lateral, the compressed root should be decompressed by enlarging the intervertebral foramen rather than by any attempt to remove the nuclear tissue. Appropriate physiotherapy and exercises, not only for their local but for their general effect as well should be prescribed postoperatively. Symptomatic relief is often disappointingly small in these patients. Moreover new peripheral disabilities, including causalgia (see pages 201, 204 and 205) may be produced, may last a long time and may of themselves, require treatment after these cervical operations.

Radiculitis from Other Types of Compression

This practically never occurs anywhere other than in the cervical spine. The *diagnosis* prior to operation, is undistinguishable from that of a lateral rupture of a cervical intervertebral disc and is made by the same methods used in patients suspected of having the latter condition. *Pathology* Irritation, compression or interruption of a root or roots is present and is caused by the invasion of the intervertebral foramen in question by so-called "hypertrophic bony spurs" or by thickening and loss of elasticity and deposition of new fibrous tissue with or without calcification in the periparticular region. As a result, there is encroachment on the root, with compression at its point of exit from the intervertebral foramen. The *signs and symptoms* are essentially the same as those listed under *Ruptured Intervertebral Disc—Cervical*. The *treatment* is essentially the same as that recommended under *Ruptured Intervertebral Disc—Cervical* except that operation will be postponed in favor of conservative therapy for a longer time.

The Treatment of Injuries to the Peripheral Nervous System

All these patients must be hospitalized for therapy

GENERAL CONSIDERATIONS

There are certain factors that affect all attempts to repair damage to the peripheral nervous system. Perhaps the most important ones are an appreciation of the source of the blood supply and knowledge of the fact that the only part of a cut peripheral nerve that can be used surgically to effect functional union is the epineurium.

The Blood Supply

This is abundant with many anastomoses a considerable overlap and a good collateral circulation.²² Nutrient branches are given off and pierce the substance of the nerve trunks from superficial longitudinal vessels that lie on the surface and in the epineurium. Their elasticity is slight, however and even minimal stretching will occlude the vessels (and especially the veins) with resulting ischemia thrombosis and necrosis at any suture line. Further stretching will add necrosis with permanent loss of function of previously undamaged parts of the nerve trunk. Residual scarring in or around the nerve diminishes still further the amount of blood and hence the nourishment that reaches the axones.

The Nerve Trunk²³

The nerve trunk, the outermost sheath or covering of which is the epineurium, is made up of fascicles each of which is surrounded by its own perineurium. The fascicles consist of nerve fiber bundles interspersed with blood vessels and containing myelinated axones within Schwann sheaths. Nerve impulses travel through the axones to reach end effectors, or from the end receptors to reach the spinal cord. Growth of a nerve can take place only with the help and connivance of the Schwann cells and only toward the periphery. Any attempt to transfix a nerve trunk with the relatively gross needle and sutures used in surgical repair (as compared with the microscopic intraneural structures) necessarily leads to hemorrhage edema, destruction of axones and development of intraneural scars that effectively

block the growth of axones and Schwann sheaths with peripheral atrophy and disappearance and replacement of these structures by scar. Penetration of the axones across the line of division of the nerve is thus prevented because of their inability to find and enter Schwann sheaths. Effective functional union of a cut peripheral nerve takes place only because the axones grow toward the periphery and in their growth are guided toward the effector or receptor end-plates by empty open Schwann sheaths. In the absence of the latter the direction of growth of the axones is confused, disseminated and irregular. It is this irregular growth that manifests itself as a neuroma on the distal cut end of the proximal segment. Degenerated and fibrosed portions of axones that have been severed from their central ends, and empty degenerated and fibrosed Schwann sheaths with hematoma and scar form the glioma—commonly called neuroma—on the proximal cut end of the distal segment.

Surgical Repair^{37 38}

Successful surgical repair of a cut peripheral nerve must meet the following requirements:

- 1 The cut end of the proximal segment must be completely viable, free of scar, neuroma or hematoma and undamaged by mechanical insult. Only in this way can free peripheral growth of the axones be assured.

- 2 The cut end of the distal segment must be completely viable, free of scar, glioma (neuroma) or hematoma and undamaged by mechanical insult. Only in this way can the axones that are to grow across the gap find open Schwann sheaths to receive them and guide them on their way toward their respective receptors and effectors.

- 3 The approximation of the opposing cut surfaces must be accurate and free of hematoma. The cut surfaces must be at right angles to the long axes of both the axones and the Schwann sheaths. Approximation of the cut surfaces must be of such accuracy that there is no overlapping, bulging or outfolding at any point, and the stitches that hold the approximated surfaces in place must be under little or no tension.

- 4 The suture material must be of the smallest practical caliber non-irritating so far as possible, swaged onto one-half curved needles that are as small as possible and inserted only through the epineurium.

- 5 The earlier the repair is done provided that it meets these basic requirements the more perfect and the greater the amount of union.

- 6 There must be no wound sepsis.

- 7 The bed in which the suture line is to rest must be as free as possible from scar and granulation tissue and if possible should be in the substance of muscle, not in approximation with tendons—and in any event not directly under the cutaneous scar.

*The Optimum Time for Suture*³⁹ With these requirements in mind, it must be apparent that although immediate suture after the injury is the ideal procedure it may be impossible, impractical or unwise to attempt it

at that time Factors that enter into making the decision concerning the best time to make the repair are

- 1 The surgical cleanliness of the original wound
- 2 The efficiency of the débridement.
- 3 The amount of local bruising.
- 4 The amount of local soft tissue destruction
- 5 The thickness of the epineurium
- 6 The ability of the surgeon to recognize and remove all the damaged neural tissue in both parts of the divided nerve This will necessitate the use of a proper electrical stimulator
- 7 The length of the gap to be bridged and the resultant need and possibility of extensive mobilization or transplantation of the nerve and positioning of the adjoining joints.
- 8 The surgical equipment available and finally
- 9 The skill and experience of the surgeon in working in this special field.

If there is any doubt in regard to any of these data it is much better to postpone formal suture of the cut nerve to some time other than that of the original repair of the wound, being content either to merely approximate the cut ends or to fasten them to some fixed tissue such as bone with the idea of preventing their retraction and thus making future suture much more easy If this decision is reached, the approximating or fixation stitches should be of 0003 tantalum wire so that the suture site or the two cut nerve ends, as the case may be can be later identified by x ray and their otherwise unrecognizable retraction upward and downward verified prior to secondary suture.

Secondary Repair of the Nerve This should be carried out within three months after injury if the greatest return of function is to be expected. It may be tried up to fifteen months however Attempts at later repair promise little. In general the higher up or more proximal the injury is on the nerve the greater the need for early repair

Technical Surgical Requirements Certain technical surgical data are worth bearing in mind. To avoid future deforming scars incisions that cross the flexion creases of any joints must parallel the crease and not run in the longitudinal axis of the limb at that point. Arterial silk sutures though causing more local tissue reaction, are on the whole more satisfactory than tantalum wire of the same or smaller size with its minimal tissue reaction. The difference lies in the greater ease in handling, the greater accuracy with which the tension can be adjusted in the knotted stitch and the decreased amount of tissue damage caused by the insertion, tying and pulling through of a silk stitch unless the surgeon is using tantalum constantly The suture lines should never be wrapped.²⁸ Tantalum foil wrappings while successful in certain surgeons hands have proven unnecessary and indeed detrimental to repair when comparative series of similar cases have been analyzed. Attempts to bridge long gaps, which cannot be approximated by transplantation or branch-stripping of the nerve by stretching the proximal segment by

means of neuroma to-glioma tension sutures with immediate full flexion and later slow extension of the involved joint may be helpful, but only within narrow limits. The necessary traction is inherently dangerous because of the possibility of causing ischemia swelling, thrombosis and stretch necrosis of the stretched part and thus potentially widening rather than narrowing the gap. Other reasons for failure are fixation of the proximal segment in the postoperative cicatrix and excessive intraneural scar formation.

Grafts ²⁰⁻²³ In late repairs heterografts and homografts are useless and should not be employed. Autogenous free grafts if they are of the "cable" type and if the individual units are not too large, may prove successful but cannot be counted upon. Single autogenous free grafts are the most successful, provided that the graftor nerve is small the grafted nerve is of the same size and the surgical technic perfect. It is better but not essential, to graft sensory to sensory and motor to motor nerves. Autogenous grafts done in stages promise more success but necessitate the sacrifice of one major nerve in the extremity.

Thrombin Suture Technic ²⁴⁻²⁸ What will probably eventually turn out to be the universal method—and what is certainly at this time the most traumatic and most effective method—of uniting cut peripheral nerve ends is the so-called "thrombin-suture" technic. This was conceived by Seddon Young and Medawar developed by Singer Tarlov and Benjamin and perfected by Bateman. In its final form the prepared cut neural surfaces to be approximated are kept from rotating by two opposite radially placed sutures through the epineurium of either segment. Accurate tensionless approximation is accomplished by carefully tying these same stitches. The suture line is then completely bathed in a solution of warmed human thrombin, with the aid of an adjustable latex mold, until the thrombin is solidly clotted (a process said to take from three to seven minutes) after which the wound is closed with the usual precautions. These include care to avoid disruption of the potentially united surfaces and removal of all clots from the cut nerve surfaces, meticulous hemostasis and no drainage. Unfortunately because of lack of knowledge of its possibilities, ignorance of the technic and unavailability of the molds, this method is at present limited in its use to specialized clinics and has not spread to those nonspecialized surgical-emergency or emergency rooms where the bulk of this surgery is first seen and where this technic is most needed.

Postoperative Identification of the Integrity of the Suture Line All repairs, whether immediate delayed or late, should be marked with a single tantalum wire stitch placed at a known distance on either side of the suture line. These stitches should be inserted through the epineurium only. Frequent x-ray films visualizing these stitches should be taken during convalescence as a check on the integrity of the suture line. A separation of the two stitches, as measured in the film and compared with the operative measurement, will notify the surgeon of the need for resuture at the earliest possible moment.

Physiotherapy

This should include massage limited passive manipulation of joints, muscle setting inside casts and, most particularly galvanic stimulation of the paralyzed muscles two or three times a week. It should be started as soon as possible and continued until voluntary motion returns or until it is apparent that the repair is a failure. Ample time should be allowed for the suture line to heal firmly before extending the joint whenever positioning of the limb has been resorted to in order to bridge a gap.

Diagnosis

Rapid, sufficiently accurate preliminary determination of the actual presence of an interruption of any of the major peripheral nerves can be carried out by a minimum of simple tests as worked out by Livingston²⁹

For the arm loss of perception of pin-prick in the distal phalanges of the index finger (median nerve) and the fifth finger (ulnar nerve) the inability to extend the distal phalanx of the thumb (radial (musculospiral) nerve) inability to flex the forearm at the elbow (musculocutaneous nerve) and loss of deltoid function and inability to abduct the arm (axillary nerve). Combinations of radial median- and ulnar-nerve deficits indicate a brachial plexus interruption.

In the leg loss of quadriceps function and probable vascular injury (femoral nerve) loss of dorsiflexion of the great toe (peroneal nerve) and plantar flexion of the great toe with the motion starting from a neutral position (tibial nerve above the knee) in both instances loss of both dorsiflexion and plantar flexion of the great toe (sciatic nerve) anesthesia in the plantar area (tibial nerve below the knee)

Functional Return

Finally it must always be borne in mind that no matter how perfect the repair and the union of a severed peripheral nerve of an extremity has been the return of function whether motor or sensory will always fall somewhat short of normal.^{27 28} Satisfactory even though not perfect, recovery can be expected under such circumstances, however. Perfection of repair is also modified by the relation of the injury to the long axis of the nerve. It appears that, in general, the more proximal the injury is to the body the further from normalcy is the eventual return of function. In the arm injuries, return of stereognosis and two-point discrimination is rare and return of normal function to the small muscles of the hand is less than in those muscles whose function is less exacting. Return of function in sciatic nerve injuries is extremely poor. Moreover the surgeon and the patient must be prepared to recognize that no matter how perfect the technic, failure of functional union may still occur for unpredictable and uncorrectable reasons. They must therefore be prepared to face re-exploration of a debrided nerve as soon as failure is apparent.

SPECIFIC INJURIES

Local Trauma with Irritation and Local Swelling of a Peripheral Nerve or Nerves

The *diagnosis* is made by an appropriate history of an appropriate injury with the immediate production of symptoms of irritation, compression or loss of function of the nerve or nerves in question. The signs will usually be those of partial interruption. The *pathology* is that of a local swelling of the nerve elements within the nerve sheath which occurs with no or at most minimal, intraneural hemorrhage. Minimal permanent changes in the microscopic structures of the nerve will be present. Perineural hematomas may organize in such a way as to compress the nerve. If they are of sufficient degree and length of application, they will cause intraneural destruction that will be manifested by permanent loss of function. *Treatment* should be devoted to maintaining the paretic or paralyzed muscles in normal tonus and length and to maintaining full mobility in range and facility of all paralyzed joints by appropriate physiotherapy, supportive splints and the like. X ray therapy if carefully used, may hasten the absorption of any large perineural hematomas and thus lessen any possible perineural scarring and compression. *Recovery* should be complete.

Compressive Median Neuritis in the Carpal Tunnel

This condition has recently been brought to the attention of the profession in a number of communications of which the most recent is by George S. Phelan under the title, "Spontaneous Compression of the Median Nerve in the Wrist."²² It is also known as "tardy median palsy." The *symptoms* and *signs* are those of a slowly developing median neuritis limited to the sensory and motor distribution of the median nerve in one or both hands. The first symptoms are sensory in nature. They may develop after some such obvious cause as damage to the bony structures of the wrist but may also appear spontaneously without any known cause. If allowed to progress untreated, major disability of the hand may develop not only because of the hyperesthesia and anesthesia but also because of weakness and atrophy of the thenar muscles. The spontaneous variety takes years rather than months to develop and is very much more common in women than in men. It does not appear to have any constant relationship to occupation or to arteriosclerosis. Phelan suggests, on the basis of the type of therapy needed and the good results obtained therefrom, that an analogy between this condition and the so-called "trigger finger" and deQuervain's disease may reasonably be drawn. It appears that the fundamental *pathology* of "tardy median palsy" is compression of the median nerve where it lies in the carpal tunnel immediately deep to the carpal ligament. *Treatment* is division of the carpal ligament through a transverse skin incision near the distal volar crease of the wrist. This exposes the median nerve, its entrance into the tunnel and the carpal ligament. The ligament is slit on its ulnar side in the direction of the long axis of the arm and the tunnel thus unroofed. If the nerve is com-

stricted locally by additional contraction of its sheath the sheath should also be split in the long axis of the nerve. According to Bunnell⁴⁴ any aberrant vessel that may be present should be dissected from the nerve and placed away from it. The *prognosis* for recovery of normal sensation is good, and for the return of function of the paretic atrophied muscles, fair.

Contusion of a Peripheral Nerve or Nerves

This condition is similar to the preceding except that intraneural hemorrhages of significant degree are added to the pathology. The *diagnosis* and *pathology* otherwise are the same as noted in the previous section with the exception that the *signs* and *symptoms* are more severe and much more lasting. *Treatment* Conservative therapy should be used only for the length of time necessary to satisfy the doctor that it is effective. With failure of the symptoms to begin to subside in two or three days, or in the face of an increase in the signs and symptoms while conservative therapy is being effectively rendered, immediate exploration of the damaged portion of the nerve or nerves should be undertaken. If after the perineural hematoma has been removed and the nerve adequately exposed, the nerve itself is seen to be swollen tense and hemorrhagic and if it does not properly transmit either motor or sensory electrical stimuli through the damaged area in accordance with its function, the epineural sheath should be split for the full length of the damaged area in order to permit decompression of the intraneural elements. The opening should be covered with "fibrin foam" and, if possible, the damaged part of the nerve should be transplanted into the substance of the belly of some adjacent muscle. The greatest gentleness, minimal handling, asepsis and meticulous hemostasis are essential. No tourniquet should ever be used. Conservative therapy as outlined under the preceding section should be started at once after the operation.

Partial or Complete Division of a Peripheral Nerve

The Acute Stage

The *diagnosis* is made from the appropriate history and physical signs and symptoms of functional interruption. In particular it should always be made an absolute rule that in all incised or stab wounds of any part of the extremities, the supraclavicular region the side of the neck the lumbar paravertebral region and the buttocks, a complete and detailed report of all sensation of the skin and of the voluntary motion of all muscles below the point of injury must be put on paper while the patient is conscious and co-operative—and particularly before he is anesthetized. This is especially necessary in injuries in which there has been extensive damage to any tendons and in injuries of the palm of the hand the wrist and the antecubital and popliteal spaces. The *pathology* is that of a partial or complete division of the nerve trunk, with the resultant characteristic microscopic degenerative and early cicatricial changes above and below the point of section. The

most effective *treatment* is atraumatic, accurate end-to-end suture of normal nerve tissue at the earliest possible moment. Rotation of the cut ends in relation to each other should be as slight as possible. The suture material should be either cotton, silk or tantalum wire, and there should be no tension on the suture line. Single adjacent sutures of tantalum wire, one on either side of the suture line should be placed at a measured distance from each other through the epineurium. The wound should be x-rayed at once after dressing and at intervals during convalescence until union is assured. If a question of separation at the suture line arises later these serial x-rays will confirm or deny it. As little suture material as is commensurate with a satisfactory union should be used. It should be limited to the epineurium and should not be put through the substance of the nerve itself. The cut nerve ends must be identified by electrical stimulation prior to suturing. The suture line *must not* be wrapped. Instead, if it is at all possible, it should be buried in the belly of an adjacent muscle. If this cannot be done, careful hemostasis, critical asepsis and repair of the wound with no dead spaces and a minimum of suture material so as to produce as little scarring as possible, offers the best hope of maximal repair and minimal intraneural and extraneural scar and compression. Postoperative therapy as outlined above should be started as soon as possible and persisted in until it is evident that repair will not take place or that function is again normal.

The Chronic Stage

The chronic stage of a partial or complete division of a peripheral nerve or nerves results from one or more of the following: failure of union in a properly timed and properly performed end-to-end suture; no surgical repair of the severed nerve; compression of the nerve in external scar or callus; interruption by internal scar; mistaken suture of the cut end of the proximal part of the cut nerve to the cut end of the distal part of a tendon; a neuroma in continuity; an unrecognized unremoved residual neuroma after attempted suture; the contraction upward and downward of the distal and proximal parts of the cut nerve; the total absence, through destruction of a large segment of the nerve, the compression and functional interruption of the nerve by application of Cargile membrane, sections of veins or arteries, fibrin films, cellophane and tantalum or other metal foil that has been wrapped about the suture line in an otherwise effective repair. *Pathology* There is scarring, with necrosis and destruction of the various intraneural structures, neuroma formation on the proximal end and a glioma on the distal end. Peripheral axonal growth is blocked. Schwann sheaths are filled or destroyed and their cut ends may be distorted or compressed. These changes may extend for long distances proximally and distally. The *diagnosis* is made from the history and physical findings, bearing particularly in mind the points listed above. The absence of signs of returning function within no more than three months of an otherwise properly performed suture in which localizing tantalum sutures were not used is

sufficient to justify re-exploration and resuture unless the original wound has been septic. In that case the wait should be prolonged until it is certain that all residual bacteria in the wound and any chance of secondary infection arising in the second operation from a nidus in the scar of the first wound have been eliminated. *Treatment* Once the decision has been reached that there is no chance of re-establishment of functional continuity in the injured nerve and once the surgeon has satisfied himself that the conservative treatment outlined under the previous headings will be of no avail in restoring innervation to previously anesthetic and paralyzed parts, and provided that chance of secondary sepsis from a previously infected wound has been estimated as a calculated risk there is no point in delaying re-exploration any longer. The operation should, first of all, be so designed that normal uninvolved nerve tissue is exposed and visible both above and below the injured area. Once normal nerve tissue has been demonstrated, the nerve trunk is followed toward the center from both ends until the damaged part is isolated and identified as far as possible. Electrical stimulation is an essential help during this dissection.

With the injured area isolated the method of repair will depend on the findings. All external compressive agents must be removed and the nerve decompressed from the outside. Compression of the intraneural elements by scar within the sheath as evidenced by gross appearance and interference with electrical conductivity can be dealt with in certain cases by the forcible injection of isotonic salt solution beneath the epineurium (internal neurolysis). If this fails and there is no expectation of any practical return of function the scarred area should be resected until normal nerve tissue is reached and an end-to-end anastomosis carried out at that point. This will undoubtedly involve the mobilization of both segments of the nerve, with freeing and mobilization of any various branches. In partial sections the uncut intact portion must not be disturbed. It should be allowed to fold on itself to compensate for the shortening necessary to effect end-to-end suture of the divided segment. Fixation of the arm or leg in such positions as will yield most slack and the transplantation of nerves from behind to in front of joints, as well as shortening of appropriate bones, are other helpful measures. When large defects make repairs otherwise impossible the two segments of the cut nerve can be sutured by their scarred or neuromatous ends to tendon or periosteum across the front of a joint in the flexed position in such a way as to produce stretching of the trunks as the joint is slowly extended. After it has been fully extended, enough extra length may sometimes be obtained to allow for end-to-end suture of normal nerve tissue across what looked at first like a completely unbridgeable gap. This is of uncertain value however because excessive stretching causes advancing intraneural destruction.

Neuromas and gliomas that are present, whether in continuity or as terminations of cut nerves, must be resected to a normal appearing cut end of the nerve before any end-to-end suture should be attempted. The same

precautions relative to suture that were noted under *Partial and Complete Division—Acute Stage* must be observed. If possible, all nerve suture-lines should be buried in the belly of any adjoining muscle. Cable-grafts made from normal uninterrupted nerves taken from the legs or leg will sometimes be successful in bridging large gaps that cannot otherwise be crossed. The cable must be of the approximate size of the host. Heterografts and homografts will not survive and are useless. Again the union of the grafts must be made end-to-end to normal nerve tissue and without tension. It should be buried in a muscle belly. The hemostasis must be meticulous and the asepsis beyond question and no such repair should be undertaken without using an adequate electrical stimulator. The *postoperative care* will depend on the difficulty of the repair. Repairs that depend for their success on the stretching of normal nerve tissue through extension of flexed joints must not be endangered by extending the joints too soon. Two weeks at least should elapse before extension is started—and more if it is thought necessary. X-ray check of the radiopaque sutures set on either side of the suture line should be made frequently to make sure that the suture line has not given way and the ends separated. Muscle-setting exercises within the cast should be practiced and physiotherapy as outlined above, started as soon as possible—and as soon as one can be sure that the end-to-end repair will not be endangered. This should be continued until maximum return of function has been demonstrated or until it is evident that the repair has failed and another operation is required.

Peripheral Nerve Injuries Associated with Fractures of the Long Bones

Certain fractures of the long bones—and especially of the humerus and the fibula—may produce loss of function of peripheral nerves that manifests itself slowly and does not become apparent until considerable time has elapsed. The neural symptoms are caused by compressing or stretching the nerve trunk on or over bone, the original injury often not damaging the nerve at all.

Fractures of the Humerus

The characteristic fractures of the humerus that cause delayed symptoms of peripheral nerve damage are those of the shaft at the junction of the middle and upper thirds in the immediate vicinity of the radial (musculo-spiral) groove and of the external condyle during childhood.

FRACTURE OF THE SHAFT The fracture of the shaft may so stretch the radial nerve by the growth of a callus or by malunion that its function is gradually interfered with to the point where a wrist-drop develops. This is particularly true when a plaster-of-paris cast has had to be applied to the whole arm and wrist to maintain satisfactory positioning of the fracture line. The nerve is almost never included in the callus—it is stretched over it and usually fixed by perineural scarring of the soft tissues. It may be thinned out or symmetrically enlarged and harder than normal. Transmission of electrical impulses through the damaged area will vary from normal

to complete absence in accordance with the severity and with the length of time the stretching has been present. *Treatment* must include resection of the callus sufficiently to relieve the stretch the transplantation if possible of the injured part of the nerve to a new bed in the belly of a muscle external and if necessary and indicated, internal neurolysis, and resection and end-to-end suture of the nerve trunk. If the condition is recognized and treated early the prognosis for recovery is good otherwise the prognosis varies with the length of time the nerve has been kept on stretch.

FRACTURE OF THE EXTERNAL CONDYLE Fracture of the external condyle during childhood leads to alteration of the comparative rates of growth of the two humeral condyles. The damaged external condyle grows little if any whereas the undamaged internal condyle grows at a normal rate. The result is that the transverse line of the elbow joint is twisted in such a way as to put the ulnar nerve on stretch in its ulnar groove every time the elbow is flexed. Depending on the degree of joint distortion, this constant local trauma and stretching will produce symptoms of partial interruption of the ulnar nerve in a varying number of years. These symptoms will usually be confined to the hand and will be both motor and sensory. The *treatment* must include exposure of the nerve at the elbow at least, with determination of the amount of interruption by electrical stimulation above the point of damage. The internal condyle of the humerus can then be resected without opening the joint cavity and the nerve left in place if a proper bed can be made for it. It must not be left in contact with cancellous bone or allowed to cross a bony prominence. If these provisions cannot be met, the nerve must be transplanted to the front of the arm well toward the center and at least the damaged area buried in the bellies of the muscles. To do this adequately the skin incision must be lengthened both upward and downward. The nerve itself should be dealt with locally by external or internal neurolysis or both but should not be resected and resutured at this time. The appearance of the nerve at the point of injury will be the same as that of the radial nerve described in the preceding section.

Fractures of the Head and Neck of the Fibula

Fractures of the head and neck of the fibula, and especially those of the neck of this bone are prone to produce some degree of common-peroneal neuritis. Even if the bone injury itself has not caused direct injury to the nerve it may nevertheless be so compressed by the application of a cast or tight bandage as to lead to severe paralysis. Indeed, even in the absence of fibula fracture the improper application of a cast to the lower leg, for what ever purpose will very frequently cause peroneal pressure neuritis. The symptoms commonly are first noticed when the cast is removed, and unless a careful neurologic examination has been made prior to its application and the nerve-function found to have been intact at that time it will be impossible to state the time of onset of paralysis. As the result of such pressure or local injury from accident, the patient develops a foot drop with paralysis of the peroneal and extensor muscles of the foot and toes. There will also be

a small area of hypesthesia or anesthesia on the dorsum of the foot. Depending on the level of nerve division, the muscular paralysis may be dissociated with retention of eversion, and the anesthesia may be extended up the lateral aspect of the lower leg. The pathology may vary from anatomic division with tearing of the ends to an intraneural neuritis with swelling, hardness and immobility or flattening and stretching of the nerve. The detailed pathological diagnosis must await confirmation by electrical stimulation. Treatment will vary with the pathology response to stimulation and gross appearance at operation. Accordingly it will vary from resection and suture to internal neurolysis, either of which may if necessary be supplemented by the removal of the necessary part of the fibula.

Neuromas

In certain instances (as, for example, in amputations) where it is desirable to eliminate neuromas, it is well to have a method of doing so that is simple and that will always succeed. Such a method was first brought to the attention of the surgical profession by Major R. C. Elmslie and by Professor McMurray then of Sir Robert Jones clinic in Liverpool.

Elimination by Method of Treatment

ELMSLIE AND McMURRAY'S METHOD.⁴⁰ The nerve to be cut is crushed in a strong clamp. If—as, for example, with a large nerve such as the sciatic—it is too large to be crushed easily it can be split in two parts each of which is dealt with as though it were an entity of itself. The nerve is then ligated at the crushed area with braided silk. The silk will vary in size in accordance with the size of the nerve being ligated but it will be classifiable as “large” under all circumstances. The nerve is then divided through uncrushed nerve tissue distal to and far enough from the tie to leave a definite fringe. The amount of crushing and the firmness of the ligature must be so adjusted as to leave the epineurium intact. If this is properly done a cone shaped closure of the cut nerve-end will form, within which the axones will be unable to grow and form another neuroma. The ligature must be non-absorbable, and the amputation of the neuroma or the ligation of the freshly cut nerve-end must be so situated as not to be included in any surface scar.

BOLDREY'S METHOD.⁴¹ Another equally satisfactory but more time-consuming and less applicable method is to bury the proximal cut end of the nerve in the medullary cavity of an adjoining bone. This was devised by Boldrey.

TENEFF'S METHOD.⁴² Teneff, of Turin, Italy has described a method in which the cut end of the proximal segment of nerve is sutured end-on in the belly of a muscle. He reports good results.

Plexus Injuries

Lumbosacral Plexus

The chances of occurrence of an accessible injury of the lumbosacral plexus in which the associated injuries were not of themselves fatal must

be small. So far as I know no report of injury to this structure *per se* is included in the wartime medical literature. Moreover I have never seen one in an active traumatic clinic during the past twenty years and I know of none reported from any other civilian clinic. It is conceivable that a stab or gunshot wound could produce an isolated reparable injury of this plexus, however. If so the *diagnosis* would have to be made as usual from the signs and symptoms of an injury to more than one peripheral nerve of the pelvis and leg, and the *pathology* would be in accordance with the kind distribution amount and length of time the damage was present before being exposed by operation after the injury. *Treatment* would be governed by the same principles that govern treatment of *Brachial Plexus Injuries*. These are detailed below.

Brachial Plexus

GENERAL CONSIDERATIONS. In the past it has been customary to identify brachial-plexus injuries as being of Erb's or Klumpke's type. The former type described damage to the upper (fifth and sixth cervical) roots, cord and branches and the latter comprised injuries to the lower (seventh and eighth cervical and first thoracic) roots, cord and branches. The medial cord was now classed with one type and again with the other. More detailed and careful study demonstrates that such classifications are artificial and of no practical use. They should be abandoned in favor of descriptions of the specific root, cord or branch damage as evidenced by consideration of all the signs and symptoms presented by the given patient. In addition to the usual contusions and stab and gunshot wounds to which other peripheral nerves are subject, the brachial plexus is peculiar in that it is most frequently subjected to tearing and stretching injuries.

CONTUSION OF THE PLEXUS. Contusion of the plexus results from a direct blow on the supraclavicular region in the presence of a minimal amount of (or no) stretching of the plexus by forcible separation of the head and one shoulder. The *diagnosis* is made from the history with the finding of little or no supraclavicular hematoma, moderate swelling of this region and a variable but usually mild degree of sensory and motor paresis in the shoulder and arm, with an occasional extension into the hand and fingers. In the newborn this is a frequent accompaniment of breech deliveries. The chief *pathology* is perineural swelling with moderate hematoma formation and compression of elements of the plexus, and possibly some mild intraneural swelling. The upper trunk or upper two trunks are the nerves usually involved. The sensory and motor paresis or paralysis is distributed accordingly. *Treatment.* The whole arm should be splinted in abduction at the shoulder at least to the horizontal, the forearm flexed and supinated and the wrist and proximal phalanges slightly extended and maintained in this position by appropriate fixation until the supraclavicular swelling is gone and the paresis and paralysis have disappeared. In addition moist heat applied locally over the swelling and possibly some carefully given x ray therapy in the same region will usually prove useful. Massage, mobilization of the

joints through their complete range of motion and active and assisted passive motion should be given three times a week. Adults may remain ambulant if they are willing and able to wear the necessary "airplane splint" constantly. In the newborn the abduction of the arm at the shoulder and flexion of the forearm at the elbow should be maintained by fastening the wrist of the involved arm by an appropriate wristlet and sling to the head of the crib and at the same time fastening the baby in such a way that it will not crawl up and get rid of the abduction by appropriate positioning of itself. Another method is to fasten the hand and wrist of the involved arm to the top of a cap that in its turn is held in place by being made to enclose the baby's whole head and to tie beneath the chin.⁴² Usually the two methods work well in combination. X ray therapy is usually not advisable in the newborn, but all other therapeutic requirements must be met. Splints are not necessary.

PERIPLEXUS HEMATOMA. This is merely an extension of the foregoing, in the direction of greater severity. The *history*, *diagnosis* and *pathology* are the same but worse. The supraclavicular swelling is greater and will be discolored, the hematoma is larger and more extensive and the signs and symptoms are more marked. There is more paralysis and less paresis. *Treatment.* The fundamental treatment is as described under *Contusion of the Brachial Plexus* above and should be instituted at the earliest possible moment. Early x ray therapy should always be used. Adults should be confined to bed and careful watch should be maintained to be sure that actual progress toward normalcy with elimination of the signs and symptoms, is taking place.

The difficult therapeutic decision to make in this and the succeeding diagnostic classifications of plexus injuries is when to abandon conservative nonoperative therapy in favor of the more radical operative methods. Early operation presents the advantage that the hematoma is still fluid, or nearly so and can be removed at least in part, and the periplexus tissues thus subjected to an adequate toilet. Moreover if any of the plexus has been torn, opportunity is thus afforded for early end-to-end repair. This is greatly simplified by the absence of an organized scar. Postoperative wound drainage can be instituted, with the resultant reduction of wound swelling and hence reduction of compression of the plexus as a whole. The disadvantages of early operation are largely those of subjecting the patient unnecessarily to an operation in the face of inevitable healing without operation the inescapable risks of any operative procedure and the formation of a scar in already damaged tissue. On the other hand, once the optimum time has passed for operative therapy and active surgical procedures are used, much less can be accomplished than could have been done earlier—and, moreover it will be much more difficult to do even that. The plexus inherently relatively immobile as it is, will even more so by peripheral fibrosis and scarring: resection of ends must be much greater because of neuroma formation scarring, and there will be an increase of the bony structure. The struc-

tures are less easily identifiable and damage that might otherwise have been apparent and reparable will be invisible and hence remain untreated. Finally the incision must be more extensive and resection or removal of the clavicle may prove necessary whereas earlier exposure would have been adequate without this procedure.

On the whole it is my belief that it is better to operate uselessly and too early than to deprive the patient of the opportunity to recover some function that he would not otherwise have had by performing an operation too late or even failing to operate at all. In any event, if operation is to be performed it should be carried out within three months—and better within one month—of the injury. Regression or increase of signs and symptoms calls for immediate surgical interference whereas cessation of improvement with loss of forward progress calls for operative interference within one month after progress has ceased or even earlier. Later operations, that is original operations performed more than three months after the injury should nevertheless be performed in spite of the discouraging outlook, if only for greater accuracy of diagnosis, for a more realistic estimate of the possibilities of any kind of treatment and as a preliminary to making a hopeless prognosis and forcing the patient to accept tendon or nerve transplants as a last resort.

At every operation, no matter what the interval after injury electrical stimulation of the exposed neural tissues is an essential part of the data on which a decision is reached concerning the therapeutic possibilities. Except in the frankly exploratory procedures that are done late the whole plexus, from the cervical intervertebral foramina to its termination in the axilla or lower must be completely visualized and all various nervous elements stimulated before a decision can be reached concerning the best method of dealing with any pathology that is present. Roots avulsed from the intervertebral foramina cannot be repaired and it is a waste of time to suture more distal peripheral divisions whose fibers originate in the avulsed. End-to-end anastomosis of any divided cords or branches after resection of the neuroma and "glioma" back to normal axones and Schwann sheaths well as of any intervening scar tissue is the preferable procedure. However because of the inherent limitation of mobility of the structures it may be better in certain instances to have "half a loaf rather than none" and to be content with something less than a satisfactory resection in the hope that some function will materialize by way of a suture that would otherwise be impossible. All scar tissue must be removed, in any event, before any kind of repair is attempted. In certain cases in which there has been enough destruction to prevent any chance of end-to-end suture of the major nerves and there are yet minor nerves still intact that, if sacrificed, could be sutured to the proximal cut ends of the irreparably damaged major nerves, such a procedure as suggested by Lurje⁴⁴ of Moscow can be carried out at any time. He has described its use in the upper injuries of the plexus. The intact long thoracic nerve was employed to reinnervate the cut suprascapular the two anterior thoracic nerves to reinnervate the cut musculocutaneous, and

two of the radial nerve branches to reinnervate the cut axillary nerve. As he notes, this procedure is useful in the late cases as a substitute for tendon transplantations which are the only other therapeutic possibilities at this time. I see no reason why, in appropriate circumstances, such a rearrangement could not be carried out at any time.

It is frequently necessary to divide the clavicle in order to obtain adequate exposure and mobilization of the plexus and its axillary extension. Postoperative union of the divided clavicle may be difficult, incomplete and deforming. The need for immobilization of the shoulder to accomplish it may interfere with the more necessary physiotherapy that is essential for the arm. As a substitute for wiring and so forth, Elkin^{42, 46} showed that the total removal of the clavicle, which was at times necessary in dealing with lesions of the great vessels in that neighborhood, neither was deforming nor affected the function of the shoulder. Moreover it saved a great deal of convalescent care and greatly simplified the entire operation. I am convinced that where the clavicle has to be mobilized in men for the repair of brachial-plexus injuries, it can be removed entirely without hesitation. The removal should be subperiosteal, an inch of bone being left intact at either end. Removal of the clavicle has the further and, at times, greater advantage of permitting so much anteromedial transposition of the shoulder as to permit an end-to-end suture within the plexus that would be impossible with the clavicle still in place. An otherwise unbridgeable gap between the cut ends may be shortened by a centimeter and sometimes more and thus completely eliminated.

The most useful incision for exposure of the plexus is one that starts one half inch lateral to and on the level with the sternoclavicular joint and extends diagonally backward and outward across the supraclavicular portion and base of the neck. A right angled extension running along the anterior aspect of the shoulder down the anterior border of the axilla and, if necessary still further down the arm will give complete exposure with a minimal division of the structures in the neck. As noted above, in such an extensive dissection the clavicle should be removed and not replaced.

RUPTURE SHREDDING AND TEARING OF THE PLEXUS. These conditions are the aftermath of a forcible separation of the head and one shoulder or of a forcible complete or partial avulsion of the arm from the shoulder. In the newborn it is a frequent accompaniment of difficult breech deliveries. The amount of damage and the degree of involvement of the plexus will depend chiefly on the degree of the lateral flexion of the neck and the simultaneous depression of the shoulder. The greatest injury will be in the upper portion of the plexus that is closest to the spine but there may be other injuries that will affect to a varying degree all the other parts of the nerves. The diagnosis will, in general, be made when the signs and symptoms that affect the region of the shoulder and upper arm have a root distribution peripherally and when there is a history of the characteristic type of injury. Symptoms of injury to the other roots and the middle and lower cords will be present in the hand and forearm. Injury to the first thoracic root or nerve

will frequently produce a Horner's syndrome. *Pathology* Such injuries will always be accompanied by a periplexus hematoma. In addition there will be typical pathologic changes that affect in varying degrees a varying number of all parts of the plexus. This may include complete rupture, intraneural ruptures, intraneural hematomas, stretch necrosis, swelling within the nerve sheath and later scar formation within and around any part of or the majority of the plexus. *Treatment* is carried out in accordance with the outline given in the preceding section.

STAB AND GUNSHOT WOUNDS OF THE PLEXUS The *diagnosis* is apparent from the history. The *pathology* will be some variant of that found in the *periplexus hematomas* and in *rupture, tearing and shredding of the plexus*. In addition there will be a wound of entrance and possibly one of exit. Associated injuries to nearby bones and other structures in the region are frequently present. The *treatment* will follow the principles laid down above under *Periplexus Hematomas*. The wound must be débrided in accordance with the type and degree of bruising and infection of the wound and before either a primary or secondary suture is attempted. Complications will include serious vascular injuries, with aneurysm formation.

The Treatment of Injuries to the Autonomic Nervous System

All these patients must be hospitalized for treatment

GENERAL CONSIDERATIONS

Injuries of the autonomic nervous system produce recognizable symptoms only when the paravertebral ganglionated chains or the terminal filaments in the extremities are involved. Other portions undoubtedly are also affected but because of their close association with other central and peripheral nervous system structures that, when injured, produce such overwhelming symptoms, injuries of the autonomic system are unrecognizable. Such is the situation in relation to the cranial parasympathetic system and, for that reason no effort has been made to deal specifically with these fibers as entities. Instead, they have been ignored (except in the case of the *nervus intermedius*) in favor of a discussion of the somatic cranial nerve with which they run. On the other hand, injury to the pelvic parasympathetic fibers produces easily recognizable symptoms and signs that can and should be differentiated, both in diagnosis and in treatment, from the signs and symptoms of injury to those somatic nerves that are closely associated with them. Finally it should be remembered that in the last analysis the chief function of the autonomic nervous system is to influence the general bodily welfare through motivating smooth muscle. Vaso-spasm, vasodilatation contraction of the bladder and bowel wall and their sphincters, the activity of the sweat glands and many other similar processes control elimination, body temperature tissue nutrition in the face of local ischemia, the blood pressure responses and reproduction. It is not unreasonable to expect, therefore that this system may be subject to injury and further that if it is subject to injury the resultant changes may likewise be widespread and have more than usually deleterious effects.

THE SYMPATHETIC NERVOUS SYSTEM

Injuries of the sympathetic as opposed to the parasympathetic nervous system are recognizable largely after the alleviation of any symptoms that may be present. This is accomplished by the production of either sympathetic paralysis or changes in bodily function that are known to be mediated exclusively by this group of nerves.

Causalgia and Allied Conditions

General Considerations

Injuries that cause the diseases in this group involve the terminal filaments of the sympathetic nerves, and the symptoms are limited to the extremities. It includes a number of disease entities that are usually regarded as separate conditions. However, the greater experience that has been made possible by the large numbers of patients injured during World War II suggests strongly that they should all be regarded as varying manifestations of the single disease process implied by the term "injury to the peripheral sympathetic nervous system." The individual entities include the *Major Causalgia of Mitchell, Morehouse and Keen*⁴⁷⁻⁴⁹, *Post Traumatic Dystrophy of the Extremities (Sudeck's Atrophy, etc.)*⁴⁹, *Volkmann's Ischemic Contracture*⁵⁰, *Traumatic Arterial Vasospasm*⁵¹ and *Homans' Minor Causalgia*.⁵² Although they differ in severity and details, all these conditions occur in association with trauma to the involved extremity; all have at some time peripheral vasospasm as part of their pathology; the symptoms of all are relieved by the production of either transitory or permanent sympathetic paralysis; and the final decision as to diagnosis rests on this therapeutic test.

The causative trauma may be either extraneous or operative. The outstanding symptom is pain of a severe, constant, burning character, and the one constant sign is some degree and type of vasomotor abnormality. It seems to be reasonable, therefore, to deduce that an injury to the peripheral sympathetic nervous system is a constant starting point but that the secondary effects of this constant factor vary somewhat with each individual disease. In major causalgia, for example, there is always an injury to a major nerve of an extremity, and the outstanding symptom is always excruciating, burning pain at the tip of the limb. In Homans' minor causalgia there is no structure or tissue that is consistently injured. However, the outstanding symptom is constant pain that is less severe than but of the same general type as that in major causalgia and that is constantly associated with peripheral vasospasm. In Sudeck's atrophy there is again no structure or tissue that is always specifically injured. Vasospasm, however, is again constant, but is now associated with edema and tends to involve particular regions and to produce atrophy of periarticular tissues, including bone. In Volkmann's ischemic contracture, although the activating injury is nonspecific, the response is strictly limited to the arteries of the limb, the ischemic necrosis being a secondary phenomenon associated with spreading involvement of the collateral arterial circulation. Traumatic arterial spasm is a milder and less effective form of the more lethal condition seen in Volkmann's ischemia.

*Major Causalgia (The Causalgia of Mitchell, Morehouse and Keen)*⁴⁷⁻⁴⁹

This is the most disabling and the severest manifestation of injury to the peripheral sympathetic nervous system. The diagnosis is established by the production of sympathetic paralysis in the involved limb, with the immediate and consequent relief of the signs and symptoms that are not caused

by tissue atrophy and ankylosis. Such a diagnostic sympathectomy is obligatory when a patient states that he has had a severe injury to an extremity—most frequently as the result of a gunshot wound—which has caused, among other things, a partial interruption of one of the major peripheral somatic nerves of that extremity. This nerve injury is usually but not necessarily a partial one. It is known also however that major causalgia will occur at times in a small number of instances in connection with a total interruption of a major somatic peripheral nerve of an extremity. Any associated vascular injury will not influence the onset, course or treatment of the disease. There is at present no good evidence to show that the patient's pre-injury emotional, vasomotor or other characteristics influence in any way the post accidental occurrence of major causalgia. The patient will complain of a constant or partially remitting burning pain, limited to the distal part of the extremity and usually restricted to the fingers or palm of the hand or toes or sole of the foot. The outstanding characteristic of this pain is its immediate increase whenever the patient undergoes any emotional activity or is subjected to such stimuli as noise, bright lights, music and so forth. Moisture applied locally to the painful area, whether hot or cold, usually affords some relief as does immobility. Sleep when attained, gives marked to total relief. For these various reasons the patients will segregate themselves as completely as possible from other people. All noise, bright light and emotional upsets, become introspective, mentally dull and, except for their overweening and all-absorbing interest in their causalgia-extremity without interest in anything except keeping it moist, wrapped-up and immobile. Moreover since the extremity is hyperesthetic they will not permit any but the most sketchy local examination.

Pathology The detailed pathology is not known. However there will be the usual pathology that accompanies a severe wound of an extremity and that includes at least a partial division of at least one major somatic nerve somewhere in its course from its exit from the intervertebral foramen to its peripheral termination. In a small minority of instances the interruption may be anatomically as well as physiologically complete.

Signs In addition to the signs caused by the nerve injury there may be the peripheral signs of either vasodilatation or vasospasm. In the first instance the involved part of the hand or foot feels warm, is redder than normal and may verge on the purple. It is mottled, the skin is normal in texture and there may be either abnormal or normal sweating. In the latter instance the involved part of the hand or foot will be pale and slightly cyanosed, the skin dry and scaly or thin and shiny and the nails curved and atrophied. There is frequently an abnormal local growth of hair as well. The peripheral pulses will be normal. There seems to be some disagreement concerning the significance of skin temperature tests, oscillometric studies and so forth designed to demonstrate alterations in peripheral blood flow. The consensus seems to be however that what evidence there is indicates that there may be relative vasodilatation even in the face of more proximal vasoconstriction. Variations in motor and sensory paralysis of the

limb will be in accord with the location and extent of the associated peripheral nerve injury. They may be virtually impossible to elicit however because of the patient's apprehension, hyperesthesia, emotional instability and self-absorption. In patients who have gone untreated for a matter of months, local changes that of themselves produce permanent functional loss are present. These include atrophy and chronic incurable ulceration of the skin, atrophy and brittleness of the nails, osteoporosis, fixation of the joints, tendon contractures, pallor, cyanosis, coldness, local increased hair growth and local sweating of the skin. In addition, personality changes, drug addictions and actual psychosis may also be present. These phenomena will not necessarily be altered in the late cases by either diagnostic or therapeutic sympathetic paralysis.

Treatment. Once the diagnosis has been established and relief of pain and other symptoms not caused by permanent deforming tissue changes has been obtained by appropriate procaine block of either the stellate and third thoracic or the lumbar sympathetic ganglia, treatment should be continued by repeating the procaine injections *provided* that the period of relief continues to lengthen significantly with each succeeding injection. If the period of relief remains the same or decreases, the production of sympathetic paralysis by injection should be abandoned and surgical sympathectomy substituted therefor. The surgical sympathectomy must be preganglionic and must produce sympathetic paralysis not only at the site of the pain in the periphery of the extremity but also in the area that has been injured. Only in this way will complete relief be obtained. Incomplete relief after sympathectomy especially in the lumbar region, is indication for reoperation, with removal of any higher ganglia and the intervening chain as well as a search for aberrant or abnormally placed ganglia. It is well to leave a silver clip as well as a silk ligature on the stumps of all branches leading to or away from the ganglionated chain as well as on the two cut ends of the chain itself. This permits the operator to check the actual extent of his denervation by postoperative x-ray examination. Although there is no evidence that regeneration of the sympathetic chain with reinnervation of the previously causalgic part will lead to a recurrence of the causalgia it is well to be on the safe side and take pains to prevent regeneration, especially in the thoracic chain. For this purpose the two cut ends of the chain as well as the cut ends of its branches should be crushed and then ligated with silk. In addition, the ends of the chain should be turned upward and downward respectively and buried by suturing them within muscular tissue.

Almost as important as the therapy by sympathectomy is the timing of the local treatment of the associated peripheral nerve injury after causalgia has developed. Regardless of type or needs, this phase of the therapy must never be used to treat the causalgia. If it is so used it will merely make bad matters worse and be entirely unsuccessful. Moreover, it should not be attempted in any case until after the sympathectomy has eliminated the causalgia. Only then can the indicated exploratory or therapeutic surgery be performed on the damaged nerve or nerves with any hope of success and

without making the patient worse than he already is. The technic for dealing with this part of the causalgic syndrome differs in no way from that used to treat peripheral nerve injuries uncomplicated by causalgia. This has been described in detail in the appropriate sections on pages 184 and 188. Foreign bodies resting in contact with the damaged somatic nerve may be removed prior to sympathectomy, however, as this procedure will at times give relief without further surgery.

*Sudeck's Atrophy*⁴⁰

This condition is known by a variety of names, is only vaguely understood and is often unrecognized. The *diagnosis* is established by the production of sympathetic paralysis of the involved extremity and consequent relief of the pain and other symptoms and signs that have not been caused by any permanently disabling changes that have already developed.

The *symptoms and signs* are limited to the extremities, and in addition produce changes that tend to localize in the neighborhood of the joint nearest the site of injury. There will be a history of an injury that may have been mild, or of a local infection that of itself does not appear to be an adequate cause of the patient's complaints. Treatment of both these conditions will have been adequate, yet the patient will still complain. In the early stages of the disease, in addition to persistent burning pain with paroxysmal exacerbations, the extremity will be warm, there will be edema of the *subcutaneous tissues and adjoining joint capsule and the muscles* will be hypertonic. The pain tends to be rather closely limited to the site of the injury and does not tend to spread. It has been stated that at this time there is local vasodilatation. In a matter of weeks the picture changes to one of vasospasm, the skin becomes shiny, cold, atrophied and cyanotic, the edema disappears, the joint becomes stiff and a spotty osteoporosis of the bones of the extremity can be demonstrated by x ray. This osteoporosis later becomes diffuse and may occur without any associated bone injury. The pain persists, and the atrophic changes are increased by disuse. The severity of the causal injury appears to have no influence on the course of the disease, which may be extremely variable. The milder cases will be self limited and may end before bone changes appear. These and the somewhat more severe cases closely resemble the patients described by Homans as suffering from minor causalgia. At the other extreme are the more serious and persistent cases, in which the pain is much worse and in which permanent disabling tissue changes take place. These can scarcely be distinguished from the late untreated cases of major causalgia referred to above. Whereas the milder forms are accompanied by only that degree of self absorption that interferes with the performance of gainful labor by the patient, those with the most severe types will have the same kind of emotional reaction that is seen in the prolonged untreated cases of major causalgia. Such patients may demand amputation, threaten or commit suicide or require confinement. Functional and economic rehabilitation is a virtual impossibility under such circumstances. The patient's pretraumatic person

ality seems to play no part in the onset or cause of the disease. There need be no significant injury to the blood vessels, muscles or somatic peripheral nerves of the extremity to produce Sudeck's atrophy, but there must have been some degree of local trauma or infection, or both, preceding the onset.

Pathology. The specific pathology is unknown. The inference is drawn, from the success attending that type of therapy that induces sympathetic paralysis, that it is in some way concerned with the vasomotor function. The permanent atrophic changes seen in the skin, subcutaneous tissues, joint structures and bone are indistinguishable from that found and described in these tissues from other, more familiar causes, however.

Signs. In the early and mild cases the signs are those of local vasodilatation imposed on whatever signs may be present that have been caused by the antecedent trauma or infection. In the later and more severe cases the signs are those of local vasospasm with associated atrophic changes and ankylosis induced by earlier periarticular muscle spasm and edema, the edema of the joint structure, the osteoporosis and the voluntary immobility of the part. Emotional and mental changes may be added.

Treatment. The treatment is the same as that outlined under *Major Causalgia*. In addition, periarterial and perivenous sympathectomy may be tried. The main artery of the limb and the chief vessels involved in the traumatized area may be thus sympathectomized in the mild and early cases with some hope of success, but if these methods fail resort should be had at once to the paravertebral procedure.

The Minor Causalgias of Humans⁴²

It is not certain that these cases are not variants of the group described above under *Sudeck's Atrophy*. Because of the frequency of their occurrence and the lack of recognition accorded them by the medical profession it seems worth-while to classify them separately, however.

The *diagnosis* is established by the production of sympathetic paralysis and resultant relief of the pain and local vasospasm in the affected limb. In most cases the symptoms and signs are limited to one extremity although a spread in milder form to the other member of the pair has been described. There will be a history of a mild injury, a prolonged rather avirulent local infection of a superficial wound, or a preceding obstructive thrombophlebitis. None of these conditions will appear to be an adequate cause for the patient's symptoms. Treatment of them will have been competent but will have afforded no relief from what appear to be atypical symptoms and signs. The patients usually are not studied from the point of view of their local vasomotor function until some months or more have passed. At that time vasospasm of the injured member with resultant cold, sweaty, cyanotic, shiny and perhaps edematous or atrophic skin will be present. It is possible that there may have been earlier local vasodilatation that was unobserved or unrecognized, exactly as been described in the *Sudeck's Atrophy* cases. Just as this local vasospasm with its deleterious effects is the outstanding sign, so a persistent burning pain that tends to be localized to the damaged

area and is made worse by cold, dryness jarring and attempted use of the part is the outstanding symptom of this condition. Homans states that "if such persons have any characteristics in common, it is the possession of cold, damp cyanotic hands and feet, as exhibitions of an underlying sympathetic irritability of central origin." These stigmas are not a prerequisite of minor causalgia, however. Many patients have a "trigger point" that when stimulated will cause an exacerbation of their pain. The secondary effects of the vasospasm that is, skin atrophy changes in the nails and subcutaneous edema tend to be closely limited to the region surrounding the site of the original injury. The pain is severe but not so severe as to produce the emotional or psychotic disturbances that are seen in major causalgia. Nor do these patients withdraw themselves from social contacts. On the other hand gainful labor is impossible because of physical inadequacy even though to a careless observer the symptoms may all appear to be subjective. Although hysteria and neurosis may be superimposed on the underlying physical defect, these patients always ask for nothing more than relief with the resultant ability to return to work, and should never be regarded as either malingerers or pure neurotics. Certain patients will show signs, such as osteoporosis and joint changes, that make their classification in the minor causalgia category rather than in Sudeck's atrophy group a matter of personal preference on the part of the surgeon. There can be no doubt that there is a large overlap between the classifications. In the patients with typical minor causalgia there need be no significant injury to major nerves or vessels of the extremity moreover the signs do not need to group themselves in relation to a joint, nor is there necessarily any significant injury to bones, muscles or tendons.

Pathology The pathology is unknown. However inasmuch as relief is afforded by the production of sympathetic paralysis it may be presumed to be connected in some way with the vasomotor functions of the extremities.

Signs In addition to the remaining signs of a pre-existing mild injury infection or thrombophlebitis of an extremity (and usually of a part of the foot or hand) the signs are those of moderately severe local vasospasm, with its associated moisture, coldness pallid cyanosis, shiny tight appearance of the skin and atrophy to a varying degree of the nails and tips of the fingers and toes. There is moderate hyperesthesia but no anesthesia and no voluntary motor paralysis. Voluntary limitation of motion may be present, however. These signs are not necessarily distributed in accordance with any anatomic or physiologic pattern. In some cases there may be bone atrophy or osteoporosis demonstrable only by x-ray examination. Search should be made for signs of a sympathetic-nervous-system lability in the uninvolved extremities.

Treatment Treatment is the same as that outlined under *Sudeck's Atrophy*. In addition if the "trigger area" is discrete and stimulation of it consistently effective, Novocain or procaine may be injected subcutaneously throughout its extent as another method of treating this particular subgroup of patients. If it is unsuccessful or only partially successful, this maneuver

should be promptly abandoned in favor of the more direct attack on the sympathetic nervous system as outlined in the two preceding sections

4. Volkmann's Ischemic Paralysis

This condition is still generally and wrongly believed to be associated with supracondylar fractures of the humerus and to have been caused by ischemia arising out of pressure from a clot in the antecubital fossa. Foix⁵⁰ however in a well documented paper has pointed out that ischemia with its resultant deformities similar to the Volkmann type is not necessarily limited to severe injuries. It certainly is not restricted to an association with fractures and may appear in the leg although it is rare for any part other than an arm to be affected. As will be seen his concept predicates a different approach to therapy and promises less invalidism and deformity than the treatment based on the older ideas.

Diagnosis The name "Volkmann's" is limited to this condition when it occurs in the arm. The diagnosis of acute Volkmann's contracture is made when the patient develops vasospasm of the periphery with cold, cyanotic, moist fingers and hand within the first thirty six hours after any type of injury. As this progresses pain of burning character accompanied by flexion of the fingers appears. This pain is increased by forcible extension of the digits. Pulsation will be absent in the terminal arteries of the extremity. In patients seen later on in the course of the disease there may be a history of constriction by a splint, circular cast or bandage with beginning tendonous contractions and continued and rather more widespread peripheral evidence of vasospasm associated with pain. Still later the circulation will have again returned to normal, but there will be permanent deformities with tendon contracture, loss of voluntary motion and muscular atrophy. The pain will have ceased by this time.

Pathology The fundamental pathology seems to be a post-traumatic segmental arterial spasm of the main artery of the limb which rapidly spreads to include the collateral circulation of the same limb with resultant ischemia of the bellies of the flexor muscles of the arm and forearm. If this process is not interrupted in time it leads to necrosis, fibrosis and atrophy of the muscles, as well as tendon contracture with deformity. The pathology is characteristic and does not resemble degeneration from sepsis or muscular denervation. There may be hemorrhage under the bicipital fascia in addition, but this is not an etiologic factor. The somatic nerves are not injured in the typical case. There may be local visible or invisible injury to the arterial wall, but this is not essential to the production of this type of spreading vasospasm.

Signs The early signs are those of peripheral painful vasospasm extending upward from the tip of an extremity towards its attachment to the body. They are accompanied by flexion contraction of the fingers and at times by some local swelling of the arm. As the disease progresses the vasospasm, and with it the pain, decreases. The flexion deformities however increase with them appear muscular atrophy paralysis and a rapidly in

creasing loss of function. With the cessation of the vasospasm the peripheral pulses return to normal.

Treatment The most important thing about the treatment is to start it at the earliest possible moment. Once the diagnosis of Volkmann's contracture has been made the patient must be considered to be as much a surgical emergency and as badly in need of instant therapy as, for example, one with a ruptured abdominal viscus. All circular and constricting dressings should be completely removed. If the elbow has been held in flexion it must be extended. The part should be elevated and kept mildly warm. These however are subsidiary procedures, are not curative and serve only to occupy the time while preparations are being made to provide specific therapy. This therapy is the production of sympathetic paralysis in the involved extremity. This should be done by the injection of Novocain or procaine about the second and third thoracic sympathetic ganglia—and, if necessary the stellate ganglion as well—on the same side as the affected arm. If the injection is properly placed and relief is to be obtained by this method, it will be apparent at once by the disappearance of the pain, the appearance of flushing, dryness and warmth in the previously cyanosed, wet, cold extremity and by the patient's ability to extend fully and painlessly his previously flexed immobile fingers.

If immediate complete relief does not follow paravertebral injection as described above and the surgeon is confident that his injection has been technically perfect, he should plan to immediately expose the brachial artery from the middle of the upper arm through the antecubital fossa to beyond its bifurcation. If after such exposure and a wait of five minutes thereafter the artery remains contracted and nonpulsating in any part, that contracted spastic portion should be excised *in toto* if it is less than 6 cm. long. If the spastic portion is more than 6 cm. long it is necessary to remove only a 5-cm. section of the spastic artery. The two free ends of the artery are ligated in either event and need not be joined. It is not necessary to expose or touch the nerves nor to incise the bicipital fascia except to expose the artery. At the close, the wound is sutured in layers that include the incised fascia. The future treatment is carried out in accordance with the other indications arising out of the injury. Treatment of the later signs and symptoms after the circulatory deficiency has repaired itself unaided are orthopedic in nature and include tendon lengthening, tenolysis, arthrodesis and so forth as indicated.

Traumatic Arterial Vasospasm⁶¹

Probably this should not be classed as a separate entity except as a means of calling attention to its importance. It lies somewhere between Sudeck's atrophy and Homans' minor causalgia.

Diagnosis Pathology Signs It is characterized by the signs and symptoms of a peripheral vasospasm of a moderate degree. In its characteristic form this is limited to the distribution of a peripheral terminal artery as, for example the dorsalis pedis or the ulnar or radial arteries. The condition is

more prone to develop in the less severe injuries that do not necessarily include direct damage to either the arteries or the nerves of the involved extremity. The pain, surface hyperesthesia, self imposed personal isolation and immobilization (usually in flexion) of the involved extremity all are characteristic of major causalgia, except that every sign and symptom is very much less in degree than is true of the more severe condition. The diagnosis is confirmed by relief of symptoms following the production of sympathetic paralysis by adequate paravertebral lumbar or thoracic sympathetic block.

Treatment Once the diagnosis is made treatment is continued by repeated injections with Novocain or procaine as long as the periods of relief continue to lengthen. If they remain the same or decrease, the injections should be abandoned and a preganglionic surgical sympathectomy should be performed. The thoracic operation should include division of all preganglionic central connections of the second and third thoracic ganglia, section of the ganglionic chain below the third thoracic ganglion and transplantation of the otherwise intact chain after ligation of its cut end, upward into muscle. In addition, the cut ends of all neural structures should be ligated with silk ties, both proximally and distally. This helps to prevent later regeneration. Any necessary lumbar ganglionectomy done for vasospasm in the legs should spare the first lumbar ganglion in the male in order to preserve his ability to ejaculate. Failure to obtain relief by this method of treatment should lead to prompt exposure of the main artery of the limb and resection of any permanently spastic segment as described under *Volkman's Contracture*.

Phantom Members

GENERAL CONSIDERATIONS Much is unknown about the causation, pathology and treatment of phantom members, but they all have one thing in common. If they develop at all it is only when the amputation has been performed after the sensory cortex has become sufficiently mature for the patient to have developed a consciousness of the living behavior of the amputated member. In connection with this, it is said that phantom limbs are not found in "infants who have had a congenital amputation or in those who have undergone amputation in infancy." On the other hand the incidence given for the occurrence of a phantom varies from 100 per cent (Foerster)²² to three out of eighty-six amputations (Cieslak and Stout).²³ Only one of this latter group had a neuroma. Livingston²⁴ described phantoms as being present thirty three and thirty-eight years after amputations done at the ages of nine and twelve years. The majority of writers place the occurrence of a phantom member at virtually 100 per cent of all amputations done within the developmental limits described above. The phantom member although usually associated with amputation of an extremity need not necessarily be so. Heuser²⁵ has described one patient whose penis was amputated and who has had a phantom penis ever since. Successful therapy will depend on a successful analysis of the symptoms or complaints that bring

the patient to the surgeon for treatment. This is always difficult and may often be impossible. The mere presence of a phantom member uncomplicated by any other symptoms, is not enough to warrant therapy. The patients so afflicted will not usually ask for treatment. If pain is added to the uncomplicated presence of a phantom member intelligent treatment with any hope of success cannot be decided upon until, if it is at all possible to do so, an analysis of the pain points to its causes or cause. Browder¹⁷ states that the incidence of painful phantom limb is 30 per cent regardless of the source of the pain.

NEUROMA GROUP Since all phantoms follow amputation there will be of necessity cut nerve ends and a scar in the stump. Depending on the method used for the handling of the proximal cut ends of the nerves, the amount of wound infection and necrosis, and the type of amputation, the proximal cut ends of the nerves will or will not grow neuromas and these neuromas will or will not be distorted and irritated by the scar. In a clean amputation with no tissue necrosis and a minimal scar there will be no pain from either distortion of the cut nerve or the presence of a neuroma *provided that* at the time of amputation *all* cut nerves are put on the stretch and crushed as high as possible without damaging the endoneurium and the nerve then tied at the point of crush by a silk tie. After ligation the nerve should be divided below the ligature and a cuff left to prevent slipping. The nerve will then retract high into the stump where it cannot come in contact with the scar and will not grow a neuroma (see page 194). However unless *all* nerves that are cut are identified and so treated, the little one that the operator forgot about *will* become adherent to the scar and *will* grow a neuroma and may be the source of pain. Cut extremity nerves not treated in that way will be sources of potential pain whether or not a phantom member is present. The pain that is thus produced tends to be local, is intermittent, occurs and increases with mechanical irritation or injury of the stump, is sharp and "electric" and may be referred out the phantom but does not originate in the ghost. A jumping stump may be associated with such pain. This kind of pain is relieved by Novocain injection of the neuroma or neuromas. It can be successfully treated by freeing *all* the neuromas from the scar, resecting them and then treating each individual resected nerve stump as described above. This therapy will relieve only this type of pain. It will have no influence on the presence of the ghost, none on vasospastic pain, none on cramping pain in the limb and none on any psychoneurosis or drug addiction that may be present. This type of pain is not relieved by any other procedure except anterolateral cordotomy done at the level of D2 segment for the lower limbs and at the level of C2 segment¹⁸ for the upper limbs. Experience and autopsy specimens have shown that high cervical cordotomy admittedly unsatisfactory as it is, permanently desensitizes to a higher level and is preferable to a medullary cordotomy for extremity pain. The thalamic cordotomy of Walker¹⁹ is still in the experimental stage. Properly performed revision and amputation of stump neuromas done on *all* nerves²⁰ and done only for the type of pain caused by

the neuroma will obviate the need of anterolateral cordotomy for this cause in almost all cases.

VASOSPASTIC GROUP Extremities that are subject to the trauma of amputation are also *ipso facto* subject to the various types of signs and symptoms described above under the headings *Major Causalgia* *Minor Causalgia* *Sudeck's Atrophy* and *Traumatic Arterial Vasospasm*. If they can be isolated and identified in these complicated cases, these symptoms and signs will correspond to those described above under these various headings. However if the pain associated with the phantom comes from several different sources in the stump the symptoms and signs will be a conglomeration of those from each separate source and therefore will be more difficult to identify. So far as the causalgia or vasospastic groups of symptoms are concerned they and they only can be verified in regard to their source by the production of a temporary sympathetic paralysis in the stump by paravertebral injections of Novocain at appropriate levels. If sufficient relief is afforded by this procedure it can be repeated or if necessary preganglionic surgical sympathectomy can be substituted for it. It is certain, however that *this* therapy will relieve *only this* type of pain. It will not alter the presence or absence of the ghost nor will it affect the syndrome of neuroma irritation the cramping pain of the ghost or any psychoneurosis or drug addiction that may be present. This type of pain is *not* relieved by any other type of procedure. However this procedure can and should be combined with any other that is appropriate in the usual complicated case.

INTRINSIC PAIN GROUP Pain other than the above in the phantom limb is, as described by Browder and Gallagher²⁷ of two varieties. In the two types considered above, any spread of the pain is toward the ghost and away from the stump. In this third group however the spread is toward the stump and away from the ghost, and the origin of the pain and its chief location are in the ghost. In the first variety of this latter group the pain is transitory with a sensation of burning, tingling, coldness, numbness, fullness or occasionally of electric shocks. It is worse immediately after amputation but for the most part gradually wears off and at its worst is no more than bothersome. It, of itself requires no treatment. It may be associated with the other types of pain described above but the therapeutic indications rest elsewhere in such circumstances.

In the other variety the pain is intractable extremely severe unremitting, a source of neurosis and drug addiction and one that urgently requires therapy. The outstanding feature is painful postural distortion of the ghost or of any part of it. It is usually a sensation of cramping, squeezing, pinching and the like. Other symptoms are the digging of the ghost fingernails in the ghost palm, the twisting backward and downward of the ghost toes under the sole of the ghost foot, a feeling as though wire were being twisted about the extremity or that it was encased in cement. All these symptoms are limited to the ghost and for the most part to the distal extremity of the phantom. They may occur in conjunction with either or both of the painful states described above but treatment of these latter does nothing to relieve

the former. Confusion on this point has "scrambled" the therapy of this type of pain. Neglect or inability to treat the pain problem of a phantom member analytically has condemned patients to unnecessary prolonged misery and invalidism. *Treatment* The suggestion by Poole⁶⁶ and Browder and Gallagher⁶⁷ that a dorsal cordotomy at the appropriate level be performed for this kind of pain appears to be well worth while, particularly as the indications are clearly stated and the results in six patients have been promising, especially as regards phantom foot. They recommend that for *lower-level distorted painful phantoms of this type* the cordotomy be performed at the midthoracic level and that all the fibers of the dorsal column on the side of the ghost be divided. For similar conditions in the upper extremity it is recommended that the lateral two-thirds of the dorsal column be divided in the level of the second cervical segment. It is said that this procedure will not disturb either gait or station. This therapy is applicable *only* to this type of pain. It will *not* relieve other types but may be combined, when necessary with other appropriate procedures. It has resulted in the loss of the phantom in addition to relief of the pain in three of the six cases.

PSYCHONEUROTIC GROUP Finally there is the group of patients who have a complicating psychoneurosis or drug addiction or both. The majority will have many operations without relief and will have gradually added to their own as yet unsolved problem by emotional instability and drug addiction. Treatment of this group is highly experimental. It is much easier to look back and say what should *not* be done in the future on another similar patient. The following can be said with some certainty however. First, no more than one revision and one reamputation of the neuromas should be done. This one that *is* done, however *must* be done properly and *must* include *all* the neuromas present in the stump. Neurotomes or other interruption of nerve trunks at higher levels, as well as reamputation of the limb for relief of pain, merely make bad matters worse. Any surgical attack on the sympathetic nervous system should be of the nature of a preganglionic operation, periarterial sympathectomy in particular being ineffective. Intrathecal injection of alcohol will do no good is dangerous and should never be carried out. Posterior rhizotomy is contraindicated, not only because it is ineffective but also because the resulting widespread peripheral anesthesia merely adds to the trouble. In particular the procedures described above should only be carried out for specific indications and never as a "last resort" gesture.

Other therapy in these patients is still highly experimental. Cortical ablation,⁶¹ which at first promised much, has proved but a weak reed to lean on. Frontal lobotomy in one of its forms is a final admission of defeat by the doctor but may yet prove to be the best solution of these difficult problems. This is especially true if the unilateral operation should prove effective. Neither lobotomy nor cortical ablation is justified, however except as a counsel of despair and all concerned must clearly understand that both

procedures as carried out in relation to the treatment of phantom members and their pains are still experimental and promise nothing.

Injury to the Central Connections of the Sympathetic Nervous System

There is suggestive evidence to the effect that extensive destructive spinal cord injuries that are severe enough to destroy all or most of the central connections of the sympathetic nervous system will produce sterility and impotence in men. There is no known treatment for this loss other than possibly artificial stimulation of the gonads in the hope of collecting enough active and viable sperm to permit artificial insemination.

There is some reason also to suppose that it is damage to the thoracolumbar chain or its central connections that renders patients with thoracic cord injuries more prone to develop bed sores than patients with cord injuries at other levels, including those who have cauda-equina injuries.

THE PARASYMPATHETIC NERVOUS SYSTEM

Injuries to the cranial portion of the parasympathetic nervous system are not recognizable as such and hence cannot be treated if and when they occur.

Injuries to the sacral portion occur almost exclusively as part of injuries to the sacral spinal cord or cauda equina. In addition to their effect on the bladder and bowel which has been dealt with under the appropriate headings in the section on spinal-cord injuries, these injuries produce impotence in men. Treatment is unavailing, except as indicated in the preceding section on injury to the *central connections of the sympathetic nervous system*. The details of the effect of such injuries in women are not known as yet. It can be stated, however, that such injuries do not affect the menstrual cycle and preclude neither pregnancy nor normal delivery of normal full term children.

The Effect of Spinal Anesthesia on the Central Nervous System

General Considerations

These patients must be hospitalized for treatment. In addition to the desired effect of analgesia or anesthesia caused by the injection of various drugs into the spinal subarachnoid space unexpected and even lethal side-effects may be caused not infrequently. In the present state of our knowledge it appears that these unexpected complications arise from toxic effects on the respiratory centers the tissues of the spinal cord and the roots of the cauda equina, or as the result of demyelination of the roots of the cauda equina by the unsuspected introduction of a strong incompatible chemical.

The Toxic Effect on the Respiratory Centers

The toxic effect of spinal anesthesia on the respiratory centers is always unexplainable and always unexpected when a spinal anesthetic is properly administered. It is recognized, however that its deleterious effect comes about directly as a result of the anoxia that it has caused and only secondarily from the toxicity. The best therapy is to keep the oxygen content of the patient's blood and tissues and especially that of the brain, from falling below a level that is incompatible with continued cell life. Measures to accomplish these ends must be initiated promptly however because three minutes of total anoxia and not more than ten minutes of serious relative anoxia is sufficient not to kill the patient but to cause such mild brain damage as to permanently decerebrate him. Coincident with the cessation of respiration during an operation that is being done under spinal anesthesia, no time should be wasted on any activity other than starting some type of efficient artificial respiration. Oxygen may be administered simultaneously but is of no value *per se* unless it can be inspired into the lungs. One fifteenth grain of strychnine and $7\frac{1}{2}$ grains of caffeine both given intravenously may be used as adjuncts to the artificial respiration but are ineffective alone. Manual artificial respiration is not efficient unless the patient can be put on the floor and two trained attendants are available to do the work. Compression and relaxation of the chest while the patient is still on the operating table produces virtually no respiratory

exchange. Immediate use of a mechanical respirator is the best insurance against destructive anoxia and anoxemia. Such an aid should be immediately available and kept constantly in proper working order. Once decerebration has been established the best that can be hoped for the patient is life as a vegetable.

The Toxic Effects on the Spinal Cord and Cauda Equina

The toxic effects of spinal anesthesia on the cord below the fourth cervical segment and on the cauda equina are also completely unpredictable. They are not recognizable until enough time has passed for the normal postanesthetic paralytic effect to have worn off at which time it is discovered that the patient is still paraplegic. Any level of the spinal cord or cauda equina may be affected but the most common site is the cauda equina.

There appear to be certain correctable factors that predispose to this paralysis. For example the incidence is very high when there is enough of a subarachnoid block to cause puddling and interference with the diffusion of the anesthetic solution. It is good prophylaxis, therefore to do a Queckstedt test before injecting any spinal anesthetic. There seems to be reason to believe that hemorrhage from a pial blood vessel may predispose to transverse toxic myelitis. It is good prophylaxis, therefore, to postpone the giving of a spinal anesthetic in the face of a bloody lumbar tap.

If a paraplegia continues after it should have disappeared, a laminectomy must be performed not later than twenty four hours after the onset of the abnormal paralysis or twenty four hours after the estimated time of recovery from the therapeutic paralysis. An occasional case will recover during this twenty four hour period. There are also other patients in whom the paralysis does not start to develop for from three to seven days there having been no abnormalities present in the interval. These cripples should be given the benefit of a laminectomy also. In the cauda-equina paralysis a lumbar laminectomy with exposure of the entire cauda should be done. The subarachnoid space must be thoroughly irrigated with sterile Ringer's solution, with the patient in a semi-erect position. After this has been done the wound, except for the dura, should be closed without drainage (see page 231). If the damage affects the spinal cord decompressive laminectomy should be performed at the appropriate level with electrical stimulation of the cord above and below the damaged area included as part of the operation (pages 228 and 231).

The Toxic Effects on Cranial Nerves

Paresis or paralysis of the sixth cranial nerve has been described as a complication of spinal anesthesia.¹⁸ It is said not to appear for seven to ten days after the anesthetic has been administered. It disappears slowly and requires no specific therapy. It is presumed to be traceable to a toxic effect of the anesthetic, but this is not certain and neither it nor any other factor has been actually demonstrated as the cause.

The Toxic Effects from Allergy or Sensitivity

Certain patients appear to be sensitive to certain of the drugs that are used for spinal anesthesia. Those that are thus sensitized may react locally in the spinal cord or cauda equina, or both in such a way as to produce an abnormal paraplegia. The most effective treatment is prophylaxis by desensitization the administration of Pyribenzamine and, best of all, the use of another kind of anesthetic. It is worth-while, therefore, to test the patient's sensitivity to the proposed spinal anesthetic before using it.

The Toxic Effects from Unsuspected Contamination of the Spinal Anesthetic

The practice of keeping sealed ampoules of anesthetic solutions that are to be used subsequently for spinal anesthesia carries certain risks with it that must be guarded against. Hairline cracks and porous glass, without dissolution of continuity in ampoules that have been stored for sterilization in solutions containing alcohol or formaldehyde are known to have led to contamination of the contents of the ampoule by these corrosive solutions. As long as the storage solution was colorless, the contamination was unknown and impossible to recognize. The injection of such a contaminated solution is known to cause a profound immediate demyelination of the roots of the cauda. If this myelin is allowed to remain floating free in the cerebrospinal fluid, it shortly causes the development of adhesions that deform and interfere with the transmission of impulses over the compressed roots. This is manifested by a peripheral sensory and motor paralysis of varying degree that is permanent, is added to the immediate post anesthetic paralysis and in the late stages is virtually untreatable. The differential diagnosis between such chemical demyelination and the toxic radiculitis described above cannot be made except at operation, and only then by the finding of free myelin or fat globules floating in the lumbar cerebrospinal fluid. The need for an early diagnostic and decompressive lumbar laminectomy is urgent and imperative in these latter cases if there is a history of a preceding spinal anesthetic induced by a solution that has been stored in a glass ampoule that has been sterilized by immersion in a solution containing alcohol, formaldehyde, an acid or a strong caustic. No delay in performing the operation should be countenanced, and the twenty-four hour wait for possible self-cure that is permissible in the former group should be abandoned. At operation an extremely thorough irrigation with sterile Ringer's solution must be carried out, with the patient in a partially erect position the dura must be left unsutured and the rest of the wound must be sutured in layers without drainage. For the late cases in which deforming fibrous adhesions are already present, a decompressive laminectomy should be performed and as much mechanical neurolysis carried out as can be done with the aid of an electrical stimulator and without producing more damage. Again it is better to leave the dura unsutured when the wound is closed.

The Storage of Ampoules Containing Solutions

The best way to treat contamination by toxic sterilizing solutions of the contents of glass ampoules is by prophylaxis. All solutions no matter what their prospective use that are bought in sterile glass ampoules may be further sterilized on the outside by storage in any satisfactory antiseptic solution provided that the solution has been colored a deep red by the addition of an appropriate inert dye. The ampoules should not be used until they have been so stored for at least thirty days and should never be used at all if on removal from the sterilizing solution they show any trace or tinge of red or pink discoloration.

Adhesive Cauda Equinalis

Although not caused directly by the anesthetic solution adhesive cauda equinalis may result from the means used to introduce it into the spinal subarachnoid space. Any lumbar puncture may produce subarachnoid bleeding. If repeated punctures are made under such circumstances a significant amount of free blood may collect. If this blood is not removed while still fluid it will organize form restricting fibrous adhesions and compress any or all of the elements of the cauda. Such compression will interfere with the transmission of neural impulses through the involved roots and a progressive peripheral loss of function will follow (See pages 63 and 72)

*Emergency Operations and Other Surgical Procedures***EMERGENCY OPERATIONS****General Considerations**

All cranial and spinal operations, whether emergency or not, carry less risk of death, prolonged illness, and permanent disabilities for the patient if they are performed in a neurosurgical clinic by a surgeon especially trained in neurologic surgery. He will have the help of an operating-room nurse and an assistant who are accustomed to neurosurgical technique. Available for his immediate use will be the consultant, x-ray laboratory and particularly blood-bank facilities that are peculiar to a large general hospital. On this account every effort should be made to transport patients needing such operative therapy to such a hospital, regardless of any reasonable distance.

It should never be forgotten, furthermore, if the question of saving time arises, that the time consumed in bringing the patient to the neurosurgeon is exactly the same or less than that consumed by the neurosurgeon in reaching the patient. From this point of view therefore transportation as such is not a factor. What is a factor however is the time consumed in the hospital preparing for and performing the operation. Provided that notification is given by telephone when the patient leaves for the hospital with a well organized neurosurgical service that neurosurgical staff, being familiar with the peculiar needs of the operation in question, having the necessary equipment and being used to dealing with such emergencies, can set up the operating room in advance of the patient's arrival. Actual experience has demonstrated that, if necessary the incision in the skin can be made fifteen minutes or less after the patient arrives at such a clinic. On the other hand, if the patient is at a hospital that has no such neurosurgical facilities, his operation must await the neurosurgeon's arrival and no significant advance arrangements can be made. Under such circumstances, moreover any necessary equipment will usually not be available in the hospital but will arrive only with the neurosurgeon the nurse and the house officer if any will be uncertain about and unfamiliar with the needs and peculiarities of the operator and, as a result, all preparations will have to be held in abeyance until his arrival. Again, experience has demonstrated that in such a case under normal circumstances the skin incision cannot be made for at least one and usually two hours after the

arrival of the neurosurgeon. This compares very unfavorably and to the patient's detriment, with the previous time of fifteen minutes.

In addition to the saving of time there is the equally important consideration of added safety for the patient. For example, a more accurate pre-operative diagnosis can be made in the neurosurgical clinic before deciding whether the operation is indeed necessary. Furthermore, if the case is one in which unpredictable emergencies may arise and have to be promptly and adequately dealt with at short notice, if the postponed operation suddenly becomes imperative or if major postoperative complications may be expected, then it is better for the patient to be situated where these problems can be dealt with most expeditiously and with greatest efficiency. These benefits more than counterbalance the disadvantages that go with increased distance from family and friends, the decreased fee that is paid the local physician and possibly even the more crowded and less familiar housing accommodations that may attend the patient's transfer. All patients with serious, doubtful, or emergency craniocerebral and spinal-cord injuries, therefore, should be transported to the nearest neurosurgical center at the very earliest possible moment, by whatever means of transportation is most easily available and regardless of any reasonable distance involved. The only contraindications to moving such a patient are surgical shock and inadequate splinting. The latter should be dealt with at the scene of the accident and must be provided before the patient may even be moved from that site. The former can be counteracted by the administration of whole blood or in its absence plasma, or failing this, 200 cc. of a 50 per cent solution of glucose given intravenously during the trip.

Even with this wide latitude however it is sometimes impossible because of fog, storm, the breakdown of public conveyances and the inaccessibility of the site of the accident, for example, to move the patient who must be operated on as an emergency to the ideal place. Rather than permit him to die by default, an attempt to perform any necessary operation must be made locally with what help and what material are at hand. The minimum equipment with the aid of which the emergency procedures described below can be performed as far as the mechanical factors are concerned is listed on page 242. Today all hospitals no matter what their type, size, kind of staff, specialty or other condition should be obligated to have such minimum equipment in their possession and usable at all times. As far as the surgical personnel is concerned, every general surgeon or other adequately trained surgeon who is on the staff of a small hospital should be able with the aid of such descriptions as are appended herewith to carry out with some degree of efficiency and in the necessary absence of skilled help those neurosurgical procedures that cannot be postponed. Such requirements are a part of the implications of the use of the word "general" as applied to surgery. He should not attempt to perform and should not be asked to perform this specialized type of surgery unless death from neglect is the only alternative but by the same token he must be able to do a better job when asked than the man without surgical train-

ing would do. Such a surgeon will be up to date on the surgical literature, will make judicious use of such postgraduate educational facilities as are available and will thus be able to meet those requirements that are necessary to make the meaning of the word "general" before the word surgery into something more than "abdominal." The responsibility for maintaining such standards rests on the staff and trustees of the hospitals.

Craniocerebral Operations

The Temporal Exploratory Trephination

This is the basic procedure for all emergency cerebral operations except those done for compound fractures. The patient is anesthetized if necessary and desirable, with an inhalation or intravenous anesthetic. The shaved scalp is sterilized. A vertical incision extending upward from the level of the external auditory meatus toward the vertex and topped by a horizontal limb that is carried 5 cm. in either direction from the end of the vertical limb is then outlined by scratches on the scalp. The upper end of the vertical limb must extend at least 3 cm. medial to the squamofrontal suture. The entire area is then circled with a 1 per cent solution of procaine, which is injected into the supragaleal tissue and the temporal muscle. Drapes are so placed as to coincide with the anesthetized circle. The vertical limb is incised first, the scalp and galea being cut in one layer and widely separated in all directions from the temporal fascia. Hemorrhage from these layers is best controlled by placing spaced hemostats on the galea, regardless of the position of the bleeders, turning the former outward and holding them in a compact group with an elastic band. The temporal fascia is then incised vertically and separated from its top attachment to the bone for 2 cm. anteriorly and posteriorly as well as from the underlying temporal muscle for its full width. The temporal muscle is incised vertically and detached on its medial end from its attachment to the bone for 2 cm. anteriorly and posteriorly as well as so widely from the bone that the squama is denuded from the region of the floor of the skull to the squamofrontal suture and for its entire lateral extent. The skin, galea, temporal fascia and muscle are then forcibly retracted laterally and held in place by a self-retaining retractor. The squama is now perforated with the Hudson drill just lateral to the squamofrontal suture and the perforation is enlarged with the other two drill points. The underlying dura is separated from the edges of the drill hole and the bony opening further enlarged with rongeurs in a circular direction, special pains being taken to remove the bony shelf at the squamofrontal suture. This opening is made big enough to permit easy insertion and later easy subdural manipulation of a medium-sized Cushing brain spoon. Care must be taken not to injure the middle meningeal vessels or, if they should be torn, to close both torn ends with silver clips or silk stitches through the dura. The dura is now opened in a stellate manner and the corners turned back over the edges of the bony opening and held in place with weighted suture loops. If possible the arachnoid should not be opened. Extradural or subdural exploration should now be

carried out over the various regions of the cerebral cortex with the aid of the brain spoon and a lighted retractor. The presence of a significant hematoma beyond the area of visualization can be determined sufficiently to justify further bone removal and a larger dural opening by retrieving significant amounts of fluid or clotted blood in the hollow of the brain spoon. Particular attention should be paid to the frontal and the parietal areas as well as to the cortex near the sagittal midline. The subdural exploration with the brain spoon must be done gently in order to avoid the rupture of any invisible bridging veins. If no clot is found extradurally or subdurally if the brain is neither swollen nor edematous and after hemostasis has been meticulous the wound including the dura, is closed without a drain. The layers are sutured separately with interrupted silk sutures inserted in the muscle, the temporal fascia, the galea and the scalp. A dry sterile dressing should be applied and held in place with a voluminous gauze bandage. The skin stitches may be removed in three or four days.

Subtemporal Decompression

If the brain is found to be or become swollen and edematous, it is essential that a large subtemporal decompression be provided. If the bony and dural openings are too small a "button" of cortex is forced strongly through this hiatus and the vessels leading to and from the "button" are compressed and closed. Thrombosis, ischemic necrosis and neighborhood swelling develop and increase. Unless they are promptly relieved they will cause herniation of the temporal cortex through the incisura, brain stem compression and death.

The operation is carried out as described above under *The Temporal Exploratory Trephination* to and including the point at which the dura has first been incised. If the cortex bulges through this first dural incision, and particularly if the former splits, the dural opening should temporarily be closed with a pad of moist cotton and more bone removed at once. To provide the necessary increase in room, the horizontal scalp incisions should be cut and the resulting anterior and posterior flaps of scalp and galea turned forward and backward. The bone removal must be carried to the point where all the squama from the floor of the skull to above the squamofrontal suture in a vertical direction and from one lateral insertion of the temporal muscle to the other in a horizontal direction is completely removed. If after this the brain is still very tight and the cortex tends to or does split, the dura should be opened by making parallel vertical incisions in it. This permits a gradual release of intradural tension, the final release being accomplished by a single incision which is carried rapidly across the artificially created dural strips. If the brain is not so tense the dura is opened in the usual stellate fashion. No attempt should be made to explore widely for subdural clots or to split the tentorium in the face of this condition, as the compression of the cortex by even a minimal amount of necessary retraction will produce ischemic necrosis and later spreading liquefaction. The brain may be needled however if there is reason to

bleeding, until the point is reached where the dura is to be opened. The diagnosis of extradural hematoma, if not already certain, will have been verified by the preliminary transtemporal exploratory trephination and the finding of a clot in the extradural space. After the opening in the bone has been made the clot is completely removed by suction except where it is adherent to any venous sinuses. A search is then made for the source of the clot. If it is arterial the open torn end of the artery should be closed with a silk suture through the dura or by a silver clip. Torn arteries will not remain closed if they are merely coagulated. If the artery is the middle meningeal and is torn too close to the foramen spinosum for the application of a clip, the foramen may be filled with and the artery closed by compression by a mixture of dry cotton and bone wax or plugged by a wooden plug whittled out of a sterile throat stick and forced into the foramen to produce the same effect. If the artery is torn outside the cranial cavity and if the bleeding cannot be permanently controlled in any other way the cranial operation must be suspended, the neck prepared and the *external carotid artery exposed and tied in the neck*.

If it is a vein that is bleeding and especially if it is the sinus that bleeds some form of Gelfoam, Fibrin foam or Oxycel should be packed over the bleeding point and held in place under firm pressure for five minutes by the clock. If none of these substances are available a muscle stamp-graft taken from the temporal muscle should be used and handled in the same fashion. Neither of the foams, nor Oxycel or muscle will control hemorrhage from a torn artery.

Any necessary enlargement of the bony opening in any direction that will allow proper visualization and control of the bleeding vessel is not only permissible but imperative. After the bleeding vessel or vessels have been closed, the dura is opened for decompression. If the compressed brain fails to expand within a reasonable time (say ten minutes) a lumbar puncture should be performed and warm sterile Ringer's solution should be injected under pressure by way of the lumbar subarachnoid space, in sufficient amounts to bring the cortex up to its proper level. The closure is carried out as described under *Subtemporal Decompression* (see page 221).

For the Removal of an Acute Subdural Hemorrhage

This diagnosis cannot be certainly made nor can the clot be lateralized, without an exploratory trephination. Preparations must therefore be made to carry out the exploration bilaterally should this prove necessary. This condition may be but usually is not such a fulminating emergency as the extradural hemorrhage. The source of the hemorrhage is always venous. The acute cases that require emergency operation do not have a significant neomembrane, are only rarely caused by the rupture of a bridging vein without associated cerebral damage, and are almost always accompanied by cerebral contusion and laceration. They are commonly found at any point over the cerebral cortex but may occur between the cerebral hemispheres or over the cerebellar cortex.

After the diagnosis has been verified by an *exploratory trephination* (see page 220) the exploration is extended to become a *subtemporal decompression* as described above (see page 221). The clots, blood or bloody fluid should all be removed especial care being taken to include those parts that lie over the tip of the frontal lobe and on the floor of the temporal fossa under the temporal lobe. Meticulous hemostasis is essential if a recurrent clot is to be avoided. Enlargement of the opening in the bone and dura to the point where proper visualization and control of any bleeding veins are obtained is therefore mandatory. Hemostasis and closure are carried out as described under *Extradural Hemorrhage* (see page 222) except that it may be necessary and wise to drain other subdural areas beside the subtemporal one. Forceful expansion of the shrunken, compressed and unexpanded brain by injection of warm sterile Ringer's solution by way of the lumbar route, as described under *Extradural Hemorrhage* (see page 222) is essential in these cases. After the wound has been closed a dry sterile dressing held in place with a gauze bandage should be applied. The skin stitches may be removed in three or four days.

For a Diagnostic Stab Wound and Therapeutic Section of the Tentorium and Incisura Tentorii

This operation is indicated within six hours after a *critical lumbar puncture* when the patient fails to improve or continues to get worse (see pages 7 and 19). It is also possible that further experience will demonstrate that it is advisable to prevent the incisural herniation as a prophylactic measure, and as a part of the operative therapy of acute cerebral extradural and subdural hematomas. If it is to be done at all it should be carried out before the brain has become so edematous as to make retraction of the occipital lobe impossible otherwise the danger of cortical liquefaction necrosis from the local pressure necessary to expose the tentorium is prohibitive.

A *subtemporal decompression* is performed as described above, and any extradural or subdural hematoma that may be present is removed. The bony opening is then enlarged posteriorly and downward sufficiently to expose the outer end of the petrous pyramid, the anterior end of the lateral sinus, its connecting group of cortical veins and the adjoining leaf of the tentorium. All further procedures are carried out posterior to the petrous ridge. An avascular area of the tentorial leaf is chosen and a stab wound $\frac{1}{2}$ to $\frac{3}{4}$ cm. long is made at this point. If there is a herniation of the temporal cortex through the incisura, cerebrospinal fluid that has been trapped beneath the tentorium will well up under pressure through this wound in amounts varying from 30 to 50 cc. With the escape of this cerebrospinal fluid, the increased intracranial pressure which until now has been only too evident, will be promptly lowered to normal or below normal levels. This permits further upward retraction of the occipital lobe during which, however great care must be exercised to avoid injury to the two groups of veins connecting the cortex with the lateral sinus. In particular the posterior

group of veins centered about the vein of Labbé must on no account be damaged or closed because it is a major channel for the drainage of venous blood from the Rolandic vein. The anterior group of the veins drain the temporal area and may in case of absolute need be divided, but at the risk of causing a permanent hemianopia. Great care must be taken to avoid mistaking an anteriorly placed posterior group for the anterior group. There is, moreover, still a third group of veins at the anterior aspect of the middle fossa that connect the Sylvian vessels with the cavernous sinus. These are not in the operative field presently under discussion. With the release of the increased intracranial pressure one blade of a scissors can be placed beneath the tentorial leaf and the leaf can be cut in an anteromedial direction through avascular areas until the rim of the incisura is reached. At this point there may be a circular venous sinus which will necessitate division of the incisura between silver clips. The incisura cannot be considered completely divided with the resulting essential relief of pressure on the brain stem unless the two separate divided portions of the incisura and their attached tentorium can be and have been folded back upon themselves—one posteriorly and one anteriorly. When this has been accomplished and meticulous hemostasis obtained, the wound should be closed as described above under *Subtemporal Decompression* (see page 221). It is essential in these cases to provide a large decompressive opening in both the bone and the dura and on no account to close the dura at the end of the operation. If there is no escape of cerebrospinal fluid through the tentorial stab wound it appears in the light of our present knowledge that it is reasonable to conclude that incisural herniation is not present and that there is therefore no need to section the incisural ring except as prophylaxis. A dry sterile dressing held in place by a gauze bandage should be applied after the wound has been closed. Stitches can be removed on the third or fourth day.

Multiple Diagnostic Exploratory Trephinations

It may be necessary at times to make multiple exploratory trephinations in order to locate an otherwise unlocalizable meningeal hemorrhage. If this procedure is determined upon it is better to reserve the subtemporal exploration over the nondominant hemisphere until the last or next to the last, the last being the suboccipital. The others should be bifrontal and placed within the hair line but anterior to the coronal suture biparietal at about the center of each parietal boss and bi-suboccipital opposite the midpoints of the underlying cerebellar hemispheres. Of the two subtemporal trephinations the right should be reserved until the last in right handed and the left until the last in left handed patients.

The whole head should be prepared and all the proposed supraoccipital incisions scratched on the scalp before any drapes have been put in place. The frontal and parietal incisions on either side should be draped in pairs and the temporal incisions covered with small sterile dressings. The wounds should be anesthetized locally with a 1 per cent solution of procaine. Their

long axis should be made in the sagittal direction. All layers, including the periosteum are cut at one time, the periosteum is scraped off the bone and the wound is held open with a self retaining retractor that includes all layers. The bone is then perforated the perforation enlarged to the full size of the largest Hudson drill point and the dura separated from the overlying bone along the edges of the trephine. A small, flat-angled dural separator should then be inserted and swung in all directions to loosen and permit the escape through the trephine of any adjacent but invisible extradural clot. The dura is then opened in a stellate manner and the same procedure carried out subdurally. In case of suspicious but uncertain findings the bony and dural opening may be enlarged with the aid of a rongeur and scissors to permit the introduction of a small Cushing brain spoon, and the explored area thus widened. If the explorations are negative the dura should but need not be closed. The soft tissues are closed with interrupted silk sutures in the galea and skin. The wounds are not drained. If the explorations have been started over the dominant hemisphere the temporal exploration should now be carried out as described, except that the incision should be vertical and placed in such a way as to form a potential part of any subsequent incision for *trans temporal exploratory trephination* as described above (see page 220). The last exploratory trephination prior to making the suboccipital incisions should be in the temporal area over the nondominant hemisphere and should, if necessary be enlarged to the point where it can have a decompressive effect. After all wounds have been closed a dry sterile dressing, held in place by an Ace type bandage should be applied. The skin stitches may be removed in three or four days.

At this point, and in the face of a negative exploration of the anterior and middle fossas, the decision must be made whether to proceed at once with the suboccipital trephinations or to postpone doing them until an estimate of the effect of any subtemporal decompression that may have been provided can be made. This will depend on the individual characteristics of the case in question. When it is decided to go ahead the patient must be carefully placed face down on a cerebellar headrest and in such a position as to insure a good airway. It is better and usually possible to do this without using an intratracheal tube which is likely to cause later respiratory complications and should be avoided except in case of absolute necessity. In any event, when the patient has been anesthetized and is in the proper position, the incisions should be anesthetized locally with a 1 per cent solution of procaine. They should be 6 cm. long, extend upward and downward and be centered over the midpoint of the underlying cerebellar hemispheres, well below the attachment of the tentorium with its contained sinuses and tributary veins. All tissues should be divided layer by layer and the wound edges held apart with a self-retaining retractor that includes all layers.

The bone should be perforated—always bearing in mind that it is extremely thin in this region—and the perforation enlarged with other drill points and rongeurs to such a size that a good exposure can be obtained

After inspection of the extradural space the dura should be opened and the subdural space inspected.

If a clot is present in either space it should be removed, and the wall of the cisterna magna should be perforated and the cistern emptied to make sure that there has been no tonsillar herniation. If necessary the openings in the bone and dura may be enlarged further in all directions to permit not only good exposure but the exercise of great care and gentleness in opening the cistern and certainly in attempting to extract any herniated tonsils. After the clot has been removed and the condition of the tonsils verified—or if the exploration has been a negative one—the wound including the dura, should be closed in layers without drainage. Interrupted silk sutures should be used throughout. A dry sterile dressing with a gauze or Ace type bandage should be applied. The skin sutures may be removed in seven to nine days if they have not caused necrosis from having been tied too tightly in which case they must be cut or removed earlier.

Débridement of Compound Fractures

COMPOUND FRACTURE OF THE VAULT *Débridement should be carried out within forty-eight hours of the infliction of the injury must be carried out within seventy two hours and should not be done until the patient is out of surgical shock and his blood pressure stabilized. The operation is much less urgent if the preliminary treatment of the scalp wound has been proper and done as described above (see page 32). The operation should not be attempted unless the preparation of the scalp is adequate unless the anesthetic is properly chosen and unless 3 pints of whole blood are available at the start.*

Under a general anesthetic preferably Pentothal sodium given intravenously with ether by the drop method on an open mask (and without nitrous oxide and if possible without an intratracheal tube) as second choice, the entire scalp should be clipped and shaved. After this has been completed the wound should be covered with dry gauze and the rest of the scalp scrubbed with gauze or a soft brush and sterile soap and water for ten minutes by the clock. The soap and water are rinsed off with three spongings with aqueous Zephuran (1:20,000) after which the scalp is scrubbed with ether on gauze three times following which all the scalp except the wound is painted with a 2 per cent alcoholic solution of iodine. The wound itself is then spread and cultured. Its edges and all the surfaces that can be reached with a cotton swab are covered with full-strength tincture of iodine. If it is available the antiseptic detergent phisoderm with 3 per cent hexa-chlorophene is to be preferred and should be substituted for the soap. There need be no consequent change in the rest of the preparation, however. Incisions that will permit débridement and adequate exposure of the damage and some method of plastic closure of the scalp are now scratched on the skin. An area enclosing these scratches and enough other tissue to allow for whatever undermining and mobilization may be necessary is encircled with

a 1 per cent solution of procaine in the subcutaneous and muscular tissues. It is unwise, unless the patient is deeply comatose to carry out the operation under the local anesthesia alone. The prepared field is draped in such a way as to expose all that lies within the procaine ring, after which the actual débridement or mechanical excision of all contaminated and non-viable tissue may be commenced. The scope of the operation will depend on the operator's judgment in regard to the extent of the contamination and the amount of what might be spoken of as any "fringe limitation" of contamination that has already developed, or in regard to the amount of control of later infection that he believes can be made good by the use of chemotherapy and the antibiotics. In this connection it should be borne constantly in mind that, except in the simplest and most superficial wounds, neither of the latter aids can be considered to be of any effect without the basic débridement. The débridement should be recognized as incomplete unless every layer that has been contaminated, from scalp to ventricle inclusive, is débrided unless all nonviable tissue is completely removed—this applying particularly to necrotic brain, blood clot and bone fragments unless the blind end of the contaminated tract is seen, débrided and emptied of all foreign bodies except for inaccessible missiles unless all large and, if possible, all dural defects are closed and unless the scalp defect over the missing bone is closed without tension and without drainage. Débridement of brain tissue should be done by suction without irrigation. A cranioplasty should not be performed at this time. Fifty thousand units of penicillin should be left in the wound at the close and the surface of the bone and the scalp tissues frosted with sulfanilamide powder. Penicillin may be put into the ventricles, but this should not be done without good reason.

COMPOUND FRACTURES INTO THE FRONTAL SINUSES. In principle, treatment by débridement is the same for compound fractures involving the frontal air sinuses as it is for compound fractures of the vault alone. There is however a variation in detail, neglect of which will lead to eventual unnecessary sepsis, morbidity and mortality. These details are covered above (see page 36).

COMPOUND FRACTURE OF THE CRIBRIFORM PLATE AND COMPOUND FRACTURE OF THE PARANASAL SINUSES OTHER THAN THE FRONTAL SINUSES. Details of operative therapy for these conditions are adequately covered above (see page 37).

Spinal Operations

Exploratory Laminectomy at other than the Cervical Level

When done as an emergency this procedure is usually designed primarily as a decompressive procedure.

The first problem is to locate the center of the incision in its proper relation to the underlying damage. Even with the help of x-rays this may be difficult because of the swelling from trauma, the amount of fat or the conformation of the patient's back. It is therefore wiser to center the incision over the upper edge of the anesthetic area and to extend the upper end at

least four spinous processes above and the lower end three spinous processes below this point. Infinitely greater harm is done the patient, when the operator refuses to provide himself with an adequate exposure than is commensurate with the increased difficulty of healing a long (as opposed to a short) wound. I prefer to have the patient on his side in the lumbar puncture position rather than flat on his abdomen. Respiratory excursions are better there is no greater danger to the spine and any inhalation anesthetic that has to be given is handled much more easily in this lateral position.

The incision should be strictly in the long axis of the spine and as accurately as possible in the midline. After the patient is anesthetized locally with a 1 per cent solution of procaine and draped, the incision is carried down to the tips of the spinous processes. Hemostasis is best obtained by placing spaced hemostats on the deep fascia without regard for the actual bleeding points, turning them backward and holding them grouped and in place with elastic bands. The superficial tissues are then separated from the lumbodorsal fascia or in the upper thoracic region, from the superficial layer of muscles for a short distance on either side of the midline. The fascia or the muscles are then cut loose from the lateral aspects of the spinous processes with the electric cutting current of the Bovie apparatus. This procedure should be started at either end of the wound, at the second normal spinous process and lamina above and below the damaged area respectively. The laminae of these same normal vertebrae are then scraped clean of muscle and the soft tissues at the two ends of the wound separated and held by self-retaining retractors. With the normal tissue planes thus demonstrated, the spinous process and laminae of the first normal vertebra above the damaged area are removed with rongeurs. This exposes normal dura, which may or may not be pulsating. This stretch of dura should then be opened in the midline and the arachnoid and the posterior surface of the cord exposed. The cord may or may not be normal, but this exposure will at least provide a plane for future dissection toward the center of the wound. If the dura has not previously been pulsating, it will start to do so with the first release of the cerebrospinal fluid from the arachnoid space, unless there is a block higher up. The same procedure is then carried out at the first normal spinous process and laminae below the damaged region. This is the point at which it is possible to determine the presence or absence of a block. If a block is present neither the dura nor the arachnoid below the damaged area will pulsate even after the fluid is permitted to escape from within the latter. By this time the surgeon should know the exact position of all the layers and structures that he must deal with. With this knowledge as a guide and by working from either end toward the center of the damaged area, he should be able to uncover the intervening spinal cord without inflicting further damage to it. Moreover if the cord is compressed at any point it should be possible to pin point the compressing agent as well as the compressed area. The surgeon will thus be certain of having decompressed the cord at the crucial point. No detailed instructions

can be given concerning the methods to be used in any individual case for these latter maneuvers, but any that are contemplated *must* be carried out gently. All tissue including the bone *must* be cut and on no account levered or pried. All scarred tissue superficial to the dura *must* be removed. At least three and preferably four spines and pairs of laminae should have been removed and the dura opened for the full length of the wound. After inspection two appropriate digitations of the dentate ligament should be cut on either side and the cord rotated alternately to both sides by traction on the dentate ligaments. This should expose the anterior wall of the spinal canal and allow the operator to satisfy himself that there has been no posterior herniation of a nucleus pulposus from an acutely ruptured disc. Any swollen area in the cord may be needled with a hypodermic needle on a syringe but should not be incised on any account. The presence or absence of a block outside the exposed area may be determined by passing a moistened No. 8 or No. 10 F soft rubber catheter through the subdural space in the appropriate direction. Preparations should now be made to *stimulate the anterior surface of the cord* if any appropriate electric stimulator is available. If normal cord has not been exposed to a sufficient extent above and below to permit this procedure another pair of laminae and a spinous process should be removed as indicated. The stimulation is first carried out below the injury. The stimulating points are placed in contact with the anterior aspect of the cord first on one side and then on the other. They must be placed inside the subarachnoid space. The current is gradually increased until there are constant and lively contractions of one or more of the leg muscles on the appropriate side in response to each stimulus. The stimulator is now moved to an analogous point above the injured area and the same current applied in the same way. If the contractions result as before the operator can be certain that the patient's cord has *not* been anatomically transected, regardless of its appearance. On the other hand, if no contractions of the leg muscles are caused by stimulation of the cord above the level of injury then the operator can be equally certain that the cord *has been* anatomically transected, regardless of its appearance, and that all chance of return of voluntary motor activity has been permanently lost.

Regardless of all other considerations no attempt should be made in the presence of a cord injury to perform an open reduction of any dislocation that may be present. The risk of producing cord damage that would not otherwise occur is too great. This, taken in conjunction with the practically constant ability of the vertebrae to grow their own solid union in these cases if kept in hyperextension over a blanket roll, and with the fact that an adequate laminectomy with a sufficiently long opening left unclosed in the dura provides relief of a cord compression for the time necessary to reduce a kyphos by such hyperextension, obviates the need for such a dangerous procedure. If there has been no cord compression the dura is closed, the suture line covered with fibrin or Gelfoam and the rest of the wound closed in layers without drainage. Interrupted silk stitches are used throughout.

Great care must be taken to place the sutures in such a way as not only to approximate the cut edges of the various layers accurately but also to close all dead spaces. Hemostasis must be meticulous. A dry sterile dressing is applied to the sutured wound and held in place with adhesive tape strips. No further dressing is necessary except in the interscapular region. Here bandages applied in such a way as to prevent forward movement of the shoulders with resultant pull on the sutured paraspinal muscles are essential to prevent disruption of the wound. They must continue to be used until the wound is completely solid—a matter of ten days at least.

Decompressive Laminectomy at other than the Cervical Level

This procedure should if possible be combined with the *exploratory diagnostic procedure* described above but may if necessary be carried out alone. This operation is done exactly as described above (see page 228) except that the dura is not sutured at the close. The space between its cut edges should be covered with sufficient fibrin or Gelfoam to insure against leakage of cerebrospinal fluid from the subarachnoid space. If this is not done a certain number of patients will develop a cerebrospinal-fluid fistula or cyst that will necessitate later reoperation and resuture.

Exploratory Hemilaminectomy in the Cervical Region

It is inadvisable and unnecessary to do a full laminectomy for exploratory diagnostic or decompressive purposes in the cervical region. Removal of the spinous processes with the interspinous ligaments, and of the lammas with the interlaminar ligaments bilaterally may so weaken the support of the head on the shoulders as to require permanent splinting that would not otherwise be needed. In particular this objection applies most strongly to the disruption of the ligamentum nuchae and its adjacent structures.

Furthermore patients with a damaged cervical spine must be handled with particular care, so far as their oxygenation and respiration are concerned. Thus their position on the operating table and the continuance of traction during the long period of necessary immobilization is of the greatest importance. The choice of anesthetic is of only slightly less importance. It is undoubtedly true that many patients have been successfully operated on in a prone position with or without traction, under an inhalation anesthetic, and have been subjected to a total laminectomy without grievous later disability but with increasing experience it has become all too obvious that to save the maximum number of lives, to cause the least possible amount of permanent disability and to accomplish the maximum good with minimal tissue damage at the time of operation, certain rules must be followed. In the first place, before a damaged cervical spine or cord can be operated on traction with Crutchfield tongs must be provided (see page 235). If this has not been done during the waiting period preceding hemilaminectomy it should be carried out as the first step in the latter operation. During transfer from bed to operating table an assistant should be assigned to do nothing except maintain traction by way of the tongs and to steady and turn the

patient's head as necessary. The patient must *never* be lifted but must always be *rolled*. The operating table must have a bar with a pulley on its far end attached to the head of the table in such a way that the traction rope leading from the Crutchfield tongs can be passed over the pulley. The patient is then placed on his side with his uppermost leg flexed at the hip and knee and his lower leg straight, the traction rope passed over the pulley and 10 or 15 pounds hung from the free end of the rope. The patient is then supported in this position with sandbags. Firm pillows are placed under his head and neck so that the cervical spine is level and both it and the head are firmly supported. He is strapped to the table in this position with his lower arm supported on an arm board. The weight of his upper body should be carried on the exact point of the shoulder with the shoulder-girdle exactly vertical, and the weight of his pelvis on the lowermost of the ilia. The body—but not the shoulders—may be rotated slightly forward if necessary but not enough to interfere with the motion of the patient's diaphragm, inasmuch as respiration depends solely on the efficiency of this muscle. Oxygen should be administered constantly. If possible the anesthetic should be limited to a 1 per cent solution of procaine injected locally. If supplementary anesthesia is necessary Pentothal sodium should be given intravenously in the protruding arm or through a leg vein. In any event, a needle sufficiently large to permit rapid transfusion of blood must be in place and connected with a constant drip of 5 per cent glucose in sterile salt solution. This apparatus *must* be kept running in constant working order. Neither morphine nor nitrous oxide should be administered at any time, but atropine in appropriate doses may be given. The second choice in anesthetics—and it is a poor one—is ether by the drop method on an open cone and *without* preliminary nitrous oxide. A mouth airway will prove helpful, but an intratracheal tube is too dangerous to use because of the probability of further cord damage through the manipulations and deep anesthesia necessary for its insertion.

The occiput and neck should be shaved and prepared as usual. The incision should be exactly in the midline and in the long axis of the neck. The correct placement of the incision will mean the difference between relatively little bleeding and a continual oozing with an attendant significant, unnecessary loss of blood. The operator's ability or inability to keep his incision in the avascular tissue spaces that separate the muscles in the midline of the deep tissues will depend on the exactitude with which the incision is located in the midline of the skin and superficial tissues. The importance of this requirement cannot be overstressed. The incision should reach from just above the posterior rim of the foramen magnum to the spine of the second thoracic vertebra. After all layers of the neck are thoroughly injected on both sides of the midline with a local anesthetic and the area draped the skin and deep tissues should be divided with a knife followed by the cutting current of the Bovie apparatus down to the tips of the spinous processes and the occipital bone. After the posterior tips of the spinous processes have been reached, the fascia covering the muscles (if it

is demonstrable) is divided lateral to the midline in such a way as to leave a fringe of this tissue, wide enough to permit its use in closing the wound, attached to the spinous processes and the interspinous ligaments. Only the upper cervical gutter is to be denuded. The muscles are then separated from the intact second and third spines and laminae above the highest and then from the two intact spines and laminae next below the lowest damaged vertebra. This should be done first with the Bovie cutting current and then subperiosteally from the side of the spinous processes and the posterior surface of the laminae as far laterally as possible. The soft tissues are separated and held in place with self retaining retractors and the first and if necessary the second intact lamina above the damaged area is removed exposing the dura. The spinous processes are *not removed* nor is the interspinous ligament cut at any point. This same thing is done at an analogous level below the damaged area. The exposed normal dura is opened above and below and the presence of an intervening cerebrospinal fluid block is demonstrated by noting subarachnoid pulsations above and none below. Pulsations above the block may not appear until after the subarachnoid space has been opened. This is a normal finding under the circumstances. The dissection now proceeds from either end toward the middle. All bone must be cut and lifted out of the wound with greatest care and should never be pried, levered or twisted as a means of removal. With the full length of dura now exposed it should be opened from end to end, great care being taken to avoid damage to the cord beneath it. This procedure should relieve any block or compression of the cord that may have been present. Two digitations of the dentate ligament are now cut and the cord is rotated gently toward the center. If more room is needed medially the base of one or more spinous processes may be removed subperiosteally and without disruption of the continuity of the interspinous ligament. The anterior wall of the cervical canal should be inspected and palpated with an angulated dural elevator to ascertain whether a nucleus pulposus has protruded. However no attempt should be made to remove the latter unless it can be lifted out easily and without any risk of trauma to the cord. Fragments from a comminuted fracture of the laminae on the other side can be reached and adjusted or carefully removed from inside the canal after subperiosteal removal of the base of one or two spinous processes opposite the damaged lamina. The cord can now be electrically stimulated as described above (see page 230) the stimulating points being placed intra-arachnoidally against the anterior surfaces first below and then above the damaged area, and the current increased until contractions of the leg muscles appear. Stimulation of the cord above the damaged area must be carried out *below* the level of the fourth cervical segment. If the cord is stimulated higher than that, the diaphragm will be made to contract forcibly and may go into spasm. With the completion of the stimulation and the relief of any cord compression, the wound is now ready to close. If the procedure has been done for diagnostic and exploratory purposes only—and this should never be undertaken as an emergency operation—the dura is closed and the suture

line sealed with a covering of fibrin- or Gelfoam. If it has been performed as a decompressive operation the dura should be left open for the full length of its incision and the space between the cut edges well covered with fibrin- or Gelfoam so that any danger of the formation of a later cerebrospinal fistula or cyst from leakage through the tear in the arachnoid is obviated. No drainage is used. Interrupted silk sutures are used to close the wound throughout. A dry sterile dressing held in place by adhesive strips is applied. No further dressing is necessary. Under no circumstances should open or closed manipulation designed to reduce any dislocation that may be present be countenanced. The risk of further damage to the cord is too great, particularly in view of the fact that such reduction can be accomplished with a minimum of risk and to a greater degree of perfection by the proper use of traction. The patient is returned to his bed with the same care and the same constant traction that was used in transporting him to the operating room. When he has been returned to his bed, his traction is re-established with the aid of the apparatus attached to the bed.

Decompressive Hemilaminectomy in the Cervical Region

The description of this operation has been included in that of *Exploratory Hemilaminectomy in the Cervical Region* (see page 231)

Débridement of Compound Fracture of the Spine and Spinal Cord

The treatment of compound fractures of the spine and spinal cord, including the cauda equina, is *not of itself an emergency*. If however a spinal-cord injury that has been compounded must be operated upon as an emergency for other reasons, the tract, including the dura, if it has been perforated, should be débrided layer by layer. The spinal cord and the cauda equina should *not* be débrided. Any foreign bodies, including those missiles that are not firmly imbedded in a vertebral body should be removed. If the dura has not been perforated, a decision whether or not to open it at the time of operation will depend on the circumstances peculiar to the particular case. In general the governing factors are the length of time since the injury, the probability of foreign bodies other than missiles being in the wound, the efficiency of the preoperative chemical and antibiotic therapy, the degree and level of cord injury as determined by clinical and x ray examinations, the possibility of dural penetration being present but invisible, the experience and skill of the operator and the adequacy of his assistance and equipment. Aside from the modification of the incision to permit excision of the tract or for a hemilaminectomy in the cervical region, and the possible failure to open the dura with a resultant inability to either visualize or stimulate the cord, the operation is carried out as described in the appropriate sections (see pages 228 and 231). These wounds should never be drained, and before being closed the raw surfaces should be frosted with sulfanilamide powder. Preoperative care so far as concerns the compounding tract should be limited to the local application of sulfanilamide

and a dry sterile dressing, with no probing or washing and a minimum of handling.

The Application of Crutchfield Type Tongs

These are built on the principle of ice tongs. A satisfactory type is manufactured with a set of drill points of such a size that the hole that is bored in the skull is of the proper caliber to receive and hold the tong-points firmly and without slipping. Traction is made from the hinge of the tongs. The tongs can be applied anywhere in the hospital and the patient should not be moved to the operating room from bed, for example, only for application of the tongs. It is important to choose the right points of insertion. These should be points that, in the anteroposterior plane will permit the tongs to exert pull in such a direction that the line of traction will be parallel to the normal long axis of the cervical spine. In the superoinferior direction the points of insertion should be so placed with regard for the width of spread of the tongs, as to be in the flattest contour of the superolateral aspects of the skull. The points should be equidistant from the sagittal line. With these two locations determined the head shaved and the skin prepared and anesthetized locally, small incisions are made in the scalp, the periosteum is incised and the drill point used to perforate the outer table. Sutures are then placed in the scalp wounds but not tied. The points of the tongs are inserted in the drill holes and the screw mechanism is operated in such a way as to force the points into the diploë and the inner table and thus to jam them tightly into the holes in the outer table. When the points have been forced in as far as they can go the instrument is locked in position and traction up to 30 pounds of weight (if necessary) is applied by means of a rope over a pulley. The technical insertion of the tongs is not difficult, but they will not function with their proper efficiency unless the drill holes—and hence the points of the tongs—are properly located on the skull. If this is accomplished and if the direction of the scalp wounds is in the line of pull, traction with this instrument will be painless.

OTHER PROCEDURES

Lumbar Puncture

(See also page 7 and *Critical Lumbar Puncture* page 19)

Lumbar puncture is a basic procedure in the diagnosis and treatment of all trauma to the central nervous system. All surgeons should be so familiar with it that they need never give a second thought to its performance.

THE EQUIPMENT The equipment needed for a lumbar puncture follows:

A 20-cc. syringe fitted with a hypodermic needle and a needle that is $1\frac{1}{2}$ to 2 inches long and that has a 20- or 22-gauge bore.

At least 10 cc. of a 1 or 2 per cent solution of procaine. If the procaine is contained in ampoules that have been sterilized by immersion and storage in sterilizing solution, they should be discarded and not used unless the sterilizing solution has been colored (preferably red or blue) and the con-

tents of the ampoules in question are crystal-clear and colorless (see page 217)

Two lumbar puncture needles either of the Fremont-Smith type or of platinum and duplicating that model

A calibrated standpipe, Fremont Smith type of manometer which is made in two sections.

Two clean dry sterile test tubes with tightly fitting rubber corks.

A pair of dry sterile gloves

Sufficient sterile gauze for sponges. Two sterile towels, ether, 2 per cent alcoholic iodine solution and a sheet folded lengthwise.

PERFORMANCE OF THE PUNCTURE The first step in the puncture will depend on whether or not the patient's co-operation can be obtained. If this is impossible or impractical the following restraint should be used. The patient should be turned on his right side one end of the folded sheet placed under his neck from behind forward, pulled across the front of his body and passed from left to right under and around both knees. This end is then brought back across the body in the opposite direction to meet the other end, which now extends forward across the left side of the neck. These two ends are twisted around each other (thus forming a figure 8 around the body—the neck being in the upper loop the knees in the lower and the crossing being opposite the abdomen) until the patient's knees and neck are sufficiently approximated to render him helpless. One attendant, either male or female then kneels on the bed (after the patient has been moved to its edge) with his or her knees firmly against the patient's abdomen and with the twisted ends grasped in both hands. There is no need to restrain the patient's arms and no need for more than one attendant if a sheet is used as described.

If the patient is co-operative no restraints or attendants are necessary or advisable. Under these circumstances the patient is placed on his right side with his back at the edge of the bed, his right leg and hip extended and his left knee and hip flexed. There should be a pillow beneath his head and his arms should be comfortably disposed. His back should be straight, and no attempt should be made to flex it.

Regardless of the co-operation of the patient the identification of the point of insertion of the needle and the skin preparation are the same, as follows. The spaces in the midline between the first sacral and fifth lumbar the fifth and the fourth lumbar and the fourth and the third lumbar spines are identified by marking each location with the operator's thumbnail. He then puts on the dry sterile gloves without preliminary scrubbing or sterilization of his hands, and without contaminating any part of the glove other than the wrist. With a sponge held in his gloved hand he now prepares, first with ether and then with a 2 per cent alcoholic solution of iodine, an area three or four inches square that is centered over the fourth and fifth interspinous space. Iodine should not be used if the preparation of the skin has involved the use of water however and it should not be used in any concentration greater than 2 per cent. It should be used as a tincture, and it may be used after acetone instead of ether. The amount of skin painted with the iodine

should be strictly limited to the local area in which the puncture is to be made. To cover the entire back with iodine or any other antiseptic as a preparation for the insertion of a needle less than 3 mm. in diameter is useless. The co-operative patient must be warned before its application that the ether will be cold. A sterile towel is now pushed between the patient and the mattress just below the sterile skin field.

The next step is to see to it that the puncture is made painless by the provision of adequate local anesthesia. If the patient is unco-operative and must be forcibly restrained as described above steps toward that end should be instituted forthwith. If he is co-operative however his continued co-operation will depend on his faith in the operator's honesty. Any statement by the operator that "there will be no pain" will, if it is not the literal truth negate all previous and future efforts to obtain such co-operation. *Per contra*, if the operator knows that he will cause pain and that it is impossible to prevent it, a statement to the patient to that effect will maintain the latter's co-operative attitude. After this preliminary any one of the thumbnail marks is picked out and an intracutaneous injection of procaine made in the midline at that point. This creates a wheal which is the future point of orientation for the insertion of the needles. The patient should be warned that the making of a wheal will be painful but thereafter there will be no pain. This future absence of pain is ensured by using no less than 10 cc. of procaine (and more if it is necessary) and by injecting each layer of tissue separately and in such a way as to form a cone of anesthetized area with its apex at the intradermal wheal and its base at the dura. The sides of the cone will include the periosteum of the adjacent bony structures, and the injected layers from the top down will be the subcutaneous tissue, the lumbodorsal fascia and interspinous ligament, the paravertebral muscles, the adjacent periosteum, the interlaminal ligaments, the epidural fat and the dura itself. After a wait of two or three minutes to give the anesthetic a chance to take effect the puncture can now be performed with the least possible reaction on the part of the patient. The needle is inserted through the skin by way of the intradermal wheal. It is pointed slightly upward in such a way as to parallel either the lower edge of the upper spinous process or the upper edge of the lower spinous process and must be kept exactly in the midsagittal plane of the body. The first obstruction will be the lumbodorsal fascia, the second the intralaminar ligament and the third the dura. Both the ligament and the dura, and especially the latter will usually give the tactile sensation of a "click." The lumbar subarachnoid space can then be entered easily and without trauma. The opposite wall of the canal should not be touched because of the danger of producing bleeding.

There is no such thing as a "dry tap" in the sense that failure of cerebrospinal fluid to pass through the needle indicates that the subarachnoid space has ceased to exist or is empty. It means rather that the operator's needle has failed to enter it. While a dry tap is practically always traceable to a misplaced needle, a very low cerebrospinal fluid pressure or a scarred adherent cauda equina with loculation of the spinal fluid may yield so little fluid as to give the impression of a dry tap. Failure of the cerebrospinal

fluid to flow adequately or steadily will mean that the needle bevel is only partly through the arachnoid or that the needle is blocked by a nerve root or has pulled out of the space. If the flow of cerebrospinal fluid suddenly stops after it has been well started, or if the pressure suddenly falls to zero, a "critical lumbar puncture" has been performed (see pages 7 and 19). Pain down the leg means that the needle has touched a nerve root. The needle should be withdrawn and reinserted. Suction should not be used to determine the location of the point of the needle. Impingement of a needle point against bone on the way in indicates failure to follow the mid-sagittal plane exactly; failure to depress or elevate the needle point so that the shaft parallels the angle of one spinous process, or failure to place the original point of the needle through the skin *exactly* in the midline. So-called hypertrophic arthritis will not, but a low surgical fusion can, prevent the doing of a lumbar puncture. Except for the psychologic implications it is as easy to do a lumbar puncture with the lumbar curve unchanged or even with the patient in opisthotonos as it is to do it with the degree of flexion that the operator imagines he obtains by the bending of the lumbar spine provided that *plenty of procaine is used and no pain is caused*. This apparent change in the position of the lumbar spine is actually produced by flexing the knees, hips and neck.

The stylette is now withdrawn the lower half of the manometer put in place and the little handle on the butt of the needle turned so that cerebrospinal fluid rises in the manometer. Air bubbles are eliminated and the assurance of obtaining the maximum rise of the column of cerebrospinal fluid is made certain by rotating the manometer to a horizontal position if necessary. The excursions of the column are then observed for their regularity, extent and coincidence with respiration. The co-operative patient can now be told that the needle is in place, that everything is satisfactory and that he should relax. After a wait sufficiently long to insure a base line of pressure that is uninfluenced by tenseness or muscular contractions, the cerebrospinal-fluid pressure is read and recorded as that figure that is the mean between the maximal and minimal excursions of the minuscus in the manometer. If fluid is to be withdrawn it should now be allowed to drip into one of the clean, dry sterile test tubes. No more fluid should be removed than is necessary for the tests that the operator has in view and certainly no more than will lower the cerebrospinal fluid pressure to a minimum of 120 mm. in a normal adult, lying on one side with his back approximately level, relaxed, comfortable and unrestrained. Unless primarily performed for special tests such as a Queckenstedt test (see below) digital decompression of the jugular veins in the neck on either one or both sides should never be done. One-sided compression is not accurate enough to justify its use. Bilateral compression is much more accurate when done as described below but there is no excuse for doing otherwise. In particular this latter procedure no matter how it is done *cannot* be countenanced in patients who are suffering from the effects of cranio-cerebral injury. No dressing is required to cover the needle puncture after withdrawal of the needle.

The Queckenstedt Test

GENERAL CONSIDERATIONS This test is designed to show whether or not there is interference with a wave of pressure flowing in the cerebrospinal fluid from the lateral cerebral ventricles to the lumbar subarachnoid space. Such a pressure wave is induced by bilateral compression of the jugular veins in the neck. This causes interference with the venous outflow from the cerebrum and the cranial chamber with a resultant increase in intraventricular pressure. The transmission of this increased pressure to the lumbar subarachnoid space is measured by changes in the column of fluid in a manometer attached to a needle that has been inserted into that space. Additional and more exact limitation of the vertical progress of the pressure wave can be accomplished by added similar observations made on a manometer that is attached either to a needle that has been inserted into the cisterna magna or to a ventricular needle inserted through posterior parietal burr holes into a lateral ventricle. Thus pressure comparisons may be made and the presence of a block between ventricles and cisternal or lumbar regions or between cisternal and lumbar regions may be determined.

THE TEST A lumbar puncture is performed as described above without the removal of any cerebrospinal fluid. The patency of the needle is verified by compression of the patient's abdomen and by the observation of a resultant and significant rise and fall of the column of cerebrospinal fluid in a manometer. The patient's neck has been previously wrapped in an ordinary Tykos sphygmomanometer arm band with the rubber bag arranged so that both jugular veins are beneath it. A blood-pressure manometer and pump are attached as usual to the cuff. After a base line of cerebrospinal-fluid pressure has been established with the cuff pressure at 0, the latter is raised by increments of 10 mm. until 40 mm. is reached and then lowered by increments of 10 mm. until 0 is reached again. Between each rise or fall a sufficient pause is made to permit the cerebrospinal-fluid pressure to level off while the cuff pressure is maintained at a constant figure. The cuff pressures are recorded in two columns, one from 0 to 40 and one from 40 to 0 and after each such reading the corresponding cerebrospinal-fluid pressure is also noted. After a base line of cerebrospinal-fluid pressure has again been established 5 cc. of cerebrospinal fluid is withdrawn and the manometrics repeated. This may be done a second time if necessary. The removed cerebrospinal fluid is carefully saved, taken promptly to the laboratory and analyzed for total protein content. This analysis is as important as the dynamics and is as much a part of the test as the latter.

A negative or normal Queckenstedt test will show a regular rapid rise of lumbar cerebrospinal-fluid pressure from 0 to 400 or 500 mm. of cerebrospinal fluid in consonance with the increase of cuff pressure from 0 to 40 and a corresponding fall to the original levels with the reduction of cuff pressure from 40 to 0. The total protein content of the cerebrospinal fluid will be less than 45 mg. per 100 cc. Abnormal tests will be indicated by an irregular slow or absent cerebrospinal-fluid pressure rise or by failure of

the cerebrospinal-fluid pressure to return to its original level at the end of the test. Any variations that may be present will be more marked in the successive tests that are done after the removal of the increments of 5 cc. of cerebrospinal fluid. Such observations justify the conclusion that there is a partial to complete block at some point between the ventricles and the manometer. In such circumstances the total protein content will be greater than normal in the specimens of fluid that have been withdrawn from the lumbar subarachnoid space.

Cistern Puncture

It is at times desirable to collect cerebrospinal fluid from, or to measure its pressure at, some point higher than the lumbar region. This should be done by a cistern puncture. In this puncture the needle is inserted through the space between the posterior edge of the foramen magnum and the lamina of the first cervical vertebra.

EQUIPMENT The equipment is the same as that described under *Lumbar Puncture* (see page 235).

PERFORMANCE OF THE PUNCTURE. The patient is placed on his right side in a comfortable position and with his head supported in such a way that the cervical spine is horizontal, fixed and supported by its right side. The head should be slightly flexed. The back should be steadied by pillows, the left leg flexed at the hip and knee and the right leg straight and extended. The puncture should not be attempted unless the surgeon has the patient's full co-operation or unless the patient is too deeply comatose to be aroused by painful stimuli. It should never be attempted without a general anesthetic on disturbed or unco-operative patients. The uppermost spinous process (usually the second cervical) that can be palpated is located and the skin over it marked with the operator's thumbnail after the posterior surface of the neck and a small area over the lowest occiput have been shaved. The field is prepared as for a lumbar puncture and the operator puts on a pair of dry sterile rubber gloves and anesthetizes the subcutaneous tissues and deeper layers from a skin wheal that has replaced the thumbnail mark. The direction of the anesthetized tract should be inward and upward and designed to terminate in the midline at the occiput just superficial to the posterior rim of the foramen magnum. The lumbar-puncture needle is then pushed through the skin with its point directed upward and inward, with the object of hitting the posterior edge of the foramen magnum. With that as a starting point, the needle is then carefully "walked" downward until it is felt to slip over the rim of the foramen. It is then pushed inward and upward for $\frac{1}{2}$ cm. the stylet removed and the needle opened. Normally by this time the operator will have felt the needle "click" through the tight dura and will have inserted the needle into the cisterna, with the resultant escape of cerebrospinal fluid. If cerebrospinal fluid does not escape, the needle may be cautiously advanced a further $\frac{1}{4}$ cm. In no case should the penetration exceed 1 cm. beyond the rim of the foramen—and even that amount is justifiable only for very good reasons. These figures should be

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reduced appropriately for the newborn and for small children. After the cisterna has been entered fluid may be collected and pressure measurements made as indicated and needed. No dressing is required to cover the needle puncture after withdrawal of the needle.

Injection and Recovery of Radiopaque Oil

These procedures can never under any circumstances be classed as emergencies. The directions herewith are only included for the sake of completeness.

The injection of radiopaque oil into the spinal subarachnoid space should be virtually restricted to the lumbar area. Cisternal injections should not ordinarily be practiced, because of the strong possibility of introducing at least some of the oil into the curve of the anterior inferior portion of the occipital bone. The oil is thus situated "around a corner" and cannot be recovered. From there it may spread through the cranial arachnoidal and ventricular spaces, with the possible production of a wide variety of organic pathology that may include complete blockage of the aqueduct of Sylvius. Visualization of the cervical subarachnoid space should be accomplished, if at all possible by moving the oil upward from the lumbar region, with the greatest care being taken to prevent any of it from entering the cranial cavity. If there is a subarachnoid block below the cervical and above the lumbar region and the need is urgent, oil may be injected into the cistern but it should be done only as a specially calculated risk.

THE INJECTION. The oil is introduced into the lumbar subarachnoid space by means of an ordinary lumbar puncture. However unless the flow of cerebrospinal fluid is completely free from interference unless the tip of the needle is entirely in the subarachnoid space and unobstructed, unless the cerebrospinal fluid is uncontaminated by blood from a hemorrhage produced by the puncture and unless the lumbar puncture is entirely painless, the oil *should not be injected*. The liability of extra arachnoidal injection of the oil with the resultant impossibility of its withdrawal and the production of scattered shadows that will complicate the interpretation of later myelograms, is otherwise too great to justify the risk. A fresh start should be made on another day. Not less than 1 cc. and preferably from 5 to 7 cc. of oil should be injected steadily and without admixture of air after which the needle should be withdrawn and the appropriate and necessary fluoroscopic observations and permanent roentgenograms made. No dressing is desirable or necessary on the needle puncture after withdrawal of the needle.

THE WITHDRAWAL. With the observations completed, the patient should again be adjusted for lumbar puncture more procaine used if necessary and the needle reinserted. After it has been ascertained that the needle tip is in the subarachnoid space and neither partially nor completely blocked, the oil should be puddled at the bottom of the sac by adjustment of the patient's body under the fluoroscope and then moved slowly upward—still under fluoroscopic control—until it is in contact with the tip of the needle. At this point suction should be exerted through the needle by a sufficiently

large syringe and the oil thus withdrawn under fluoroscopic control and, if possible without interruption. The needle is then withdrawn. No dressing is necessary over the needle puncture after the latter has been withdrawn.

Removal of a Broken Lumbar puncture Needle

A not infrequent complication of lumbar punctures is the discovery that because of a sudden movement on the part of the patient the point of the needle has been jammed into the bone the shaft twisted and the needle broken, leaving the buried piece invisible in the tissues of the back. If the stylette happens to be in place at the time of the break, the broken piece may rarely be extracted with the former when it is pulled out. If it is not in place, however it is a great mistake to try to reach the fragment by any means short of a formal operative removal.

The patient should be taken to the operating room after the point of insertion of the needle through the skin has been carefully marked. In the operating room the field should be prepared as is customary for a regular surgical operation and then, with the puncture wound as the center a circular incision with a radius of from 1 to $1\frac{1}{2}$ inches should be scratched around the wound on one side of the midvertical line of the back. The two ends of the circumferential scratch should then be extended straight laterally across the midvertebral line for a distance of 1 inch. After draping, the semicircular flap with its base between the two ends of the circumferential incision and with the puncture wound in its center should be cut in such a way as to include in one layer all the tissues of the skin and the superficial and deep fascia down to the lumbodorsal fascia. This flap, when turned laterally on its uncut base will reveal the broken end of the needle fragment in nine out of ten cases. Removal is then simply a matter of extraction of the fragment under direct vision. In the few instances in which the fragment is too short to protrude through the lumbodorsal fascia, a similar semicircular flap of lumbodorsal fascia with its base at the midvertical line should be dissected from the muscle on the side of the spinous processes that corresponds to the side of the insertion of the needle. After recovery of the fragment, the wound should be sutured in layers with interrupted silk stitches and without drainage.

If the needle is neither visible nor palpable with the aid of these exposures, the laminae will have to be uncovered just as in an ordinary laminectomy. If there is any delay between the discovery of the break in the shaft of the needle and the start of the operative removal of the fragment, adequate x ray films (preferably stereoscopic anteroposterior and lateral views) must be taken before attempting removal.

MINIMAL INSTRUMENTAL AND OTHER EQUIPMENT

Instrumental Equipment

The following list includes a minimal amount of instrumental and other equipment that is essential for the proper performance of emergency neurosurgical operations. The capital C and capital L marked before each item

indicate that it is necessary in the doing of cranial operations and laminectomies, respectively. The firm names listed are those who are either the sole manufacturers of the instrument thus designated or the distributors through whom they are sold.

Knives

C L Handles, Bard-Parker No 4 (2 each) No 7 (1 each)	3
C L Blades, Bard Parker Nos 22 15 11	Sufficient number

Forceps Hand

C L Cushing Brain Forceps, with Teeth (1 x 2)	3
C L Cushing Brain Forceps, without Teeth (Serrated Jaw)	3

Forceps to Apply Silver Clips

C L McKenzie 6-inch Curved	2
C L McKenzie, 6-inch Angulated	2
C L McKenzie Clip-Holding Rack	1
C L McKenzie Silver Brain Clips (100 to a vial)	2 vials

Scissors

C L Curved Mayo, 5 $\frac{3}{4}$ inch	1
C L Metzenbaum, 7-inch	1
C L Lahey's Goiter Curved, 5 $\frac{3}{4}$ inch	3

Hemostats

C L Halstead, Straight, Box Lock, $\frac{1}{2}$ Serrated Pointed Tips	72
C L Kelly Curved	6
C L Halstead Mosquito Snaps Curved	6
C L Allis Clamps	6
C L Lahey Goiter Clamps (Triple Hooks)	6

Retractors Hand

C Cushing, Aluminum (Mueller No NS-225)	3
C Flexible Copper Retractors (Reed No 1991) 1 2 3 and 12 inch	1 each
C Cushing Brain Spatulas (Reed No 1928) 6- 6 $\frac{3}{4}$ and 7 $\frac{3}{4}$ inch	1 each
C Cushing Brain Retractors (Reed No 1931) 8-inch	2
C L Cushing Vein Retractors (Reed No 1932) 7 $\frac{3}{4}$ -inch	2
C L Volkman's, Blunt Rake (Reed No 2160) 4 & 6 Prongs	2 each
L Israel Blunt (Mueller No GO-992) 10 $\frac{1}{2}$ inch 4 Prongs	2
C L Pilling Brain Retractor Electrically Lighted with Ex	

tra Bulbs and Dry-Cell Battery Box (Mueller No NS-P15150)

2

Retractors Self Retaining

C	Gelpi	2
C	Lurens Mastoid (Reed No 2833)	2
C	Munro (Codman & Shurtleff)	2
L	Beckman's Goltz blunt (Reed No 2838)	2
L	Chandler with All Blades (Edw Weck & Co., 135 Johnson Street, Brooklyn, New York)	2
L	Frazier Laminectomy	2
L	Kanaval Laminectomy (Mueller No P 15106)	2

Periosteal Elevators

C L	Love-Adson, Sharp 7½ inch (Mueller No NS-684)	1
L	Adson (Mueller No NS-687)	1

Rongeurs

C L	Stille-Luer Multiple Action, Angular (Reed No 1555)	1
C	Bacon Cranial Forceps, Angular 8½-inch (Mueller No NS-295)	1
C	DeVilbiss Bone Cutting, 7 inch (Reed No 1556)	1
C L	Stille-Horsley Double Action Bayonet (Reed No 1549)	1
L	Luton Bone Cutting, Angled on Side, 7½-inch (Reed No 1515)	1
L	Kerrison Punch, 8½ and 6½ inch (Reed)	1 each
L	Stille 6-inch Straight, Angled on Side, and Angled on Flat (Reed Nos 7230 7231 and 7233)	1 each
L	Pituitary Rongeur Cushing, Straight and Angled (Codman & Shurtleff)	1 each

Drill

C L	Hudson Cranial Set with Perforator Burrs B & C and Cerebellar Extension (Mahady No 1630)	1
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Miscellaneous

C L	Frazier Dural Hook	1
C L	Frazier Dural Elevator	1
L	Graham Nerve Hooks, blunt (Mueller No NS-605)	3
C L	Grooved Director	1
C L	Clinical Thermometer for Ringer's Solution	1
C L	Asepto Syringes, 2-oz.	2
C L	Suction Tips	
	Glass Drinking Tubes	2
	Frazier Metal with Finger Release Valve, sizes 1 2 and 3 (Mueller No NS-565)	1 each

C L Backhaus Towel Clips	12
C L Jones Towel Clips	12
C Frazier Ventricular Needles (Reed)	6
C L Penrose Type Drains, $\frac{3}{8}$ and $\frac{1}{2}$ inch	Sufficient
C L Novocain kit for Local Anesthesia	
10-cc. Tonsil Syringe	2
Lucr Lok Tip Needles, 3-inch (21 gauge) 2 inch (20-gauge) and $\frac{3}{4}$ -inch (25-gauge)	2 each
C L Fremont Smith Lumbar Puncture Kits Complete	3
C L Wide Rubber Bands	24
C L Large Steel Safety Pins	24
C L Luken's Bone Wax	Ample supply
C L Gelfoam H12 Thrombin 1000 U Normal Salt 20 cc Vial	Ample supply
C L Sterile Ringer's Solution	Ample supply
C L Sterile Normal Salt Solution	Ample supply
C L Sterile 1% or 2% Novocain or Procaine Solution	Ample supply
L Crutchfield-Type Tonga with Drill Point	1
L Soft Rubber Catheters No. 6 F	4
C Scissors for Cutting Hair	1
C Electric Hair Clipper	1
C L Safety-Razor Blades	12
C L Kelly Hemostats	2
C L Towels, Sterile	24
C L Half Sheets, Sterile	12
C Craniotomy Sheets, Sterile	2
L Laparotomy Sheets, Sterile	2
C L Bovie Apparatus with 2 Sets of Attachments (Knife Needle Loops etc.)	1
C L Wall or Other Suction (with 2 lengths of large, uncollapsible rubber tubing to be sterilized and included with the instruments)	1
C L Provision for prompt emergency whole-blood transfusion	Adequate
C L Champagne Cerebellum Headrest, to fit any table (No S-1580-D)	1
The following medicine should be available <i>in the operating room</i>	
1 50% glucose solution for intravenous injection	
2 Caffeine for intravenous injection	
3 Coramine for intravenous injection	
4 Neosynephrine	
5 Morphine	
6 Pentothal sodium for intravenous injection	
7 Strychnine for intravenous injection	
8 Aminophylline for intravenous injection	

Bed Attachments for Bedridden Paraplegic and Quadriplegic Patients

Some form of overhead frame is essential to the care and rehabilitation of these patients. Whatever is used should be permanently attached to the

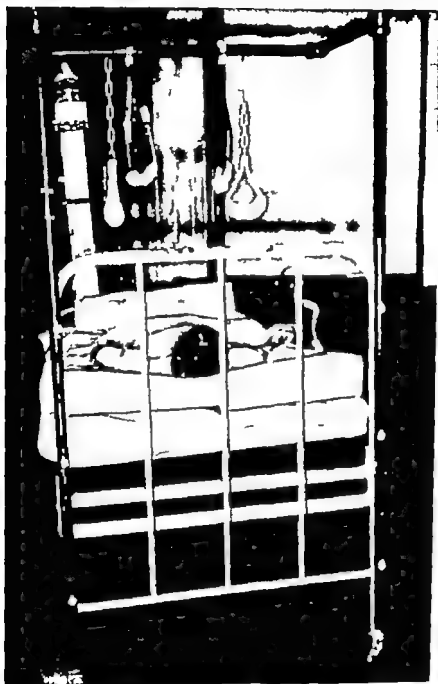


Fig. 47 Framework of iron piping bolted to the head of a bed.

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frame with appropriate attachments will do but the modification shown in Figures 10 and 47 has proven more than usually successful. It is made of 1 $\frac{1}{2}$ -inch pipe two 6 $\frac{1}{2}$ -foot lengths of which are bolted with three bolts each to the head of the bed. The tops of the two uprights are joined by a cross-bar. Projecting from just below the top of the two uprights and held by right angled pipe fittings is a rectangle of pipes. These extend toward the foot of the bed for two feet and from the cross bar are hung two chains equipped with handles and so placed as to be 9 $\frac{1}{2}$ inches toward the middle from each side-bar. This will provide firm support for any self-mobilization the patient may wish to do. In addition—and to be used as exercisers—are loops of rubber tubing fastened to the head and sides of the bed and to the cross-bar overhead. Proper use of these loops will exercise all the shoulder arm and hand muscles. Their resistance can be increased by increasing the amount of tubing in each loop.

Wheel Chair Specifications

The most useful and reliable wheel chair is the folding automobile chair made by Everest and Jennings of California. Those patients who have to use it for long periods of time should pad the seat with a square of thick sponge rubber. This is more attractive and more easily handled if it is enclosed in a waterproof plastic casing equipped with a handle. Quadriplegics may need detachable extensions for the back to support their head and shoulders and certainly should have padding on the arms. The ordinary specifications follow below.

This chair should be the Everest and Jennings universal model with the large wheels behind, should be equipped with hand rims, brakes, chain locks, detachable arms, foot board extension adjustment and adjustable foot boards with wooden leg rest panels. The small front wheels should be eight inches in diameter.

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*The Cost of Medical Care in Paraplegia
and
How It May be Modified by Rehabilitation Services*

By

STANWOOD L. HANSON Assistant Vice President
Liberty Mutual Insurance Company
Boston Massachusetts

The concept that the cost of the medical care of spinal-cord injuries may be modified by competent rehabilitation of the paralyzed patient is a new one. The practical application of this concept more than justifies its wider application. The data on which this claim is based are described in the following pages and are derived from the experiences of a large insurance carrier of workmen's compensation liability. They have been amassed in the course of dealing with the disability and the resultant cost of medical care which were associated with spinal-cord injuries that have occurred in industry. My observations also indicate that the factors so characteristic of industrial cases are not dissimilar to those arising out of similar injuries that have occurred in the armed services, from highway accidents or from athletic injuries to young people. The problems that may be anticipated by the person or organization responsible for providing the care and paying the bill are largely the same.

Under workmen's compensation laws industry and casualty insurance carriers have the responsibility for paying not only a weekly indemnity to the patient but also for any necessary surgical care, hospitalization, nursing services, drugs, prosthetic appliances and attendant care. The amounts vary and are dependent upon that particular law which is in effect in the individual state, territory or federal jurisdiction. When compensation laws were first adopted both types of payments were limited both as to amount and as to the period of time during which they would be payable. Over the years, however, constant legislative changes have increased the amount of these payments, as well as the length of time during which they must be made to cover medical treatment and hospital care. At the time of writing, for example, in twenty-one of the states, territories and federal jurisdictions,

indemnity payments continue in the event of permanent total disability for as long as the injured worker may live. Spinal cord paralysis usually qualifies as such a permanent total disability. In thirty-five of the states, territories and federal jurisdictions medical services must be paid for as long as they are deemed to be necessary even though they may be required throughout the life of the patient. In patients with spinal cord injuries these services may well include several major operations and other procedures as well as special drugs and apparatus. In addition to this special nursing may be needed around the clock for a period of weeks, months or even years. Since the cost of hospital care including extras in most well-equipped institutions exceeds \$15 per day it is a matter of easy arithmetic to determine that such services may cost, exclusive of the surgical fees from \$1,200 to \$1,400 per month per patient. This is from \$6,000 to \$16,000 per year dependent upon the amount of private nursing care required. Since indemnity benefits will usually vary from \$1,000 to \$1,800 a year in addition the over-all cost of one of these cases may well range from \$7,000 to \$18,000 per year. Any surgical fees must be added to this sum. They will vary greatly and will depend upon the amount and type of surgery required. These high costs may continue for a long time. In the files of the Liberty Mutual Insurance Company for example there exist records of cases in which hospitalization has been required for eight, ten and fifteen years. It is seldom realized that under present-day conditions the medical costs in just one of these cases may exceed \$100,000.

These high costs are in large part a new community responsibility. They are a new responsibility because prior to World War II persons who suffered from injuries of the spinal cord usually died immediately following the injury or within a matter of months. During and since World War II however surgical methods of handling these injuries have been more generally understood and hence more effective. Patients who formerly died will live and unless steps are taken to prevent it, will by virtue of that very fact suffer many complications that would not have had time to develop otherwise. This adds months, years or possibly even a lifetime of hospitalization and special nursing care.

The experiences of the armed services in World War I as compared with those of World War II illustrate this problem very well. Figures which have been quoted from armed service records indicate that there were some 400 cases of traumatic spinal paralysis as the result of World War I. Only a few of these patients were alive two years after the receipt of their injury. During and following World War II, however some 2,300 or 2,400 "paraplegics" were brought back to this country from combat areas. The great majority of these are still alive and moreover appear to have an indefinite life expectancy. The experience of the Liberty Mutual Insurance Company as disclosed in the medical histories contained in its files is similar. Prior to World War II, most of their patients with spinal-cord injuries caused by industrial accidents died within a reasonably short period following the injury. On the other hand the few who lived were forced to remain in hos-

pitals because of the complications that developed as an integral part of their longevity. Most of these invalids required special nurses. The improvement in care that developed during and after World War II with the advent of the new drugs and the spread of improved surgical methods increased their life expectancy but did not reduce their morbidity. The cost of the resultant increase in necessary hospital and nursing care therefore rose but with no compensatory increase in the rehabilitatory benefits derived therefrom. Methods adequate to correct this economic and physical wastage had still to be devised.

The present life expectancy of spinal cord paralytics is unknown because no appropriate actuarial experience has been accumulated under present day conditions but there are known to be many patients who have maintained a good health record for a number of years and whose life expectancy appears to be unchanged from normal. If these patients are to remain without benefit of rehabilitation and hence to require hospitalization for indefinite periods, the medical costs will run into tens of thousands of dollars. For example the writer knows of one patient with quadriplegia on whose account an American reinsurance company anticipates a loss that may approach \$400,000.

From observations of patients with traumatic spinal paralysis which has been caused by other than industrial injuries, the problems involved appear to be entirely similar. Unless very specialized diagnostic and surgical procedures are undertaken, the tragic and bizarre complications of "paraplegia" set in and unless methods looking toward rehabilitation are employed, hospitalization or attendant care will be required indefinitely.

It is apparent that the problem of paralysis as the result of a spinal-cord injury cannot be dealt with in part only. To do so merely emphasizes the tragedy of the disability. It has to be dealt with as a whole, not only to preserve life but to rehabilitate the individual from a pathetic invalidism and to do away with the tremendous cost of that invalidism.

Under many of our compensation laws, a person who has lost both arms, both legs, or any two thereof either by severance or by paralysis, is deemed to be suffering from a permanent total disability under the law and is entitled to the maximum of compensation benefits provided by the particular law that applies in his case. In a number of states this provision is for a lifetime. No savings in weekly indemnity can, therefore, be gained by the rehabilitation of a patient who is being paid under the provisions of such a law. Even in the absence of such legal provision and because paraplegics are not good candidates for competitive employment, the possibility of savings in indemnity is doubtful and does not constitute a principal financial goal. Any major saving will have to lie therefore, in the field of relief from the need for long-continued hospitalization, special nursing, or attendant care in a convalescent home and from the cost of treating frequent flare-ups of those complications which are more likely to occur in cases not properly motivated and not taking daily exercise. An extra dividend on the money invested in such savings will be the immeasurable satisfaction that

comes from returning one of these patients to his home and family with the prospect of a reasonably active life in his community.

In an attempt to meet these needs the Liberty Mutual Insurance Company began to study the progress being made by the few clinics or services specializing in the over-all handling of this type of case. These studies were conducted to determine whether or not there existed programs for paraplegics that would prevent the onset of many of the complications usually anticipated, and that could rehabilitate the patients to a point where the need for indefinite hospitalization and special nursing care no longer existed. It had been demonstrated in a few clinics that patients could be taught to balance themselves when sitting up, to move from bed to wheel chair and from wheel chair to bathroom stool and bath tub, to take care of their own bodily needs, to learn to walk with the aid of braces and crutches, to dress themselves, travel, drive cars and in numerous cases even to work at sedentary jobs. Thus such patients might expect to live reasonably normal lives and to avoid the necessity of permanent hospitalization—a life confined to bed and the need for nursing or attendant care.

Encouraged by these findings similar rehabilitatory procedures have been undertaken in twenty-one cases covered by this company under workmen's compensation laws. It was soon apparent that whenever such paralyzed patients could be brought together and handled in a ward or in a group of hospital rooms constituting as it were a special service with specially trained hospital nurses and attendants for them, the need for the special nurses was reduced or eliminated entirely in a matter of weeks. When to this was added the specialized care described herewith and by Dening, Deyoe and Ellison,⁴ not only was the need for special nursing care removed but the patient was stimulated to become independent and to constantly attempt to do more and more for himself. Furthermore, a comparison with our earlier experience demonstrated the value of moving such cases into specialized surgical clinics for paraplegics, where all of the diagnostic and surgical services described in this volume were applied and where a co-ordinated and all-out program for the rehabilitation of the patient was instituted. The length of hospitalization was shortened, the incidence of complications reduced to a minimum, permanent morbidity was done away with and life expectancy increased with elimination of the need for future hospitalization, attendant care at home and the like. Thus the costs were reduced at a fairly early date and were maintained at a low level as time went on, not only without detriment to the patient but usually to his considerable benefit.

These rehabilitatory efforts were successful to some degree in all twenty-one patients, all being graduated from the necessity for continuous hospitalization. Three or four in the older age group where there was a multiplicity of complications may require future hospitalization. In the balance of the cases, however, no substantial future periods of hospitalization are anticipated although they will require periodic examinations to forestall any possibility of later trouble. Fourteen of the patients have been

returned to some form of sedentary work or are engaged in their own businesses, varying all the way from repairing watches to driving a truck with special controls and conducting a trucking business. Employment is intermittent in some instances but, in all of the fourteen, the patients appear to have been permanently returned to a relatively normal family and community life.

Based upon the best possible estimate of the life expectancy of these fourteen patients and figured from the point of view of savings in both indemnity and in medical expense a study of the estimates of costs demonstrates that this rehabilitatory effort has saved the company \$903,900 00 for an actual expenditure of \$115,830 00. Forty thousand seven hundred dollars were savings in weekly indemnity payments and \$863,200 00 were savings in medical costs (Table 9). There have been no cases in which

TABLE 9 SAVINGS EFFECTED BY REHABILITATION OF FOURTEEN PARALYZED PATIENTS

	NOT REHABILITATED	REHABILITATED	SAVINGS BY REHABILITATION
Estimated weekly indemnity	\$305 700 00	\$265,000.00	\$40 700 00
Total medical cost	\$1,127 700 00	\$264,500 00	\$863,200.00
Total cost of rehabilitation		\$115,830.00	\$903,900 00

efforts at rehabilitation did not pay off to some extent, and the wisdom of applying appropriate and qualified rehabilitation services in industrial paraplegia cases seems to be well demonstrated. It would appear therefore, that not only the best method but virtually the only method which can be utilized to minimize the cost of the care of any patients paralyzed as the result of an injury to their spinal cord or cauda equina is the application of the very best possible diagnostic and surgical services at the beginning, the provision of the best available nursing care for as long as it is needed but no longer and the use of all possible procedures that lead to rehabilitation as early as the condition of the patient will permit. Only by these methods can the need for continued hospitalization or attendant care be avoided and the hope of restoring the unfortunate victim to independence and earning power be realized.

The Responsibility of the Hospital Trustees and Staff

Much still needs to be done to reduce the mortality and alleviate the invalidism and suffering that develop all too frequently as the result of injuries to the nervous system. The information necessary to accomplish this is steadily accumulating but is not readily available. This text was written to fill this gap and as an attempt to make some of this knowledge available to all who want it. The determination to translate this information into better care should have the enthusiastic support of hospital staffs and, if necessary, the active stimulus of hospital trustees. Their activities along these lines are complementary. Unilateral action by one without the support and co-operation of the other is ineffective. For example, lavish equipment provided by the trustees is not only useless but may be dangerous if the staff is not professionally competent to use it to its best advantage. The promise of good community medical and surgical care that its presence implies can not be fulfilled otherwise, and without its proper use the citizens that provide the money to pay for such equipment are deceived and deprived of the returns that they should have on their investment. *Per contra*, a competent staff that is progressive and eager to raise the standards of medical care and practice may well be frustrated and prevented from doing their best work by the absence of such equipment. In this case the community will believe that the staff are to blame when actually it is not their fault but rather attributable to the short-sighted policies of the trustees. Only by insistence on the part of the staff that the trustees provide them with adequate tools and help, and by the trustees that every member of the staff continually endeavor to improve his professional background and knowledge, will the hospital justify its support by and position in the community it is designed to serve.

The above requirements are usually easily met in so far as "general surgery" and "general medicine" are concerned. The same also applies to certain specialties such as nose and throat or genitourinary practice. The public has been educated to demand the best and to recognize incompetence in the handling of patients in these categories. Unfortunately this is not true of neurosurgery. This is a relatively new and unfamiliar subject. It is regarded with something akin to awe by the community. Moreover, the common belief by the public and medical profession alike is that the number of patients suffering from disease or injury to the nervous system is few

at the time of the original suture will be greatly handicapped and may be impossible because of the need for a wide excision of the scarred cut ends of the nerve. Even at best they are much less effective than a properly done primary suture in so far as return of function is concerned.

The other group—those patients who have been paralyzed as the result of spinal cord damage—are even more pathetic. This has been emphasized in the preceding chapter from the point of view of community costs. The picture is even worse from the point of view of community responsibility. The institutions in this country that are equipped (outside of the veterans and certain armed services hospitals) to properly care for such invalids can be counted on the fingers of one hand. The number of such patients in need of such institutional care as a necessary prerequisite to rehabilitation otherwise unattainable to them is in the many thousands. These patients require a long hospitalization; they and their families frequently either do not have or shortly run out of enough money to pay hospital bills; they tie up hospital beds that the staff wants to use for more fluid surgical or medical cases; their care requires a meticulous attention to detail which the surgeon in charge is not only unwilling to learn how to give, but will not provide even if he does know how because of the time it takes; and finally those civilian centers that are willing and able to cope with this problem are already badly overcrowded and have waiting lists. What more natural, then, than for the community hospital that has had this incubus dumped in its lap and for the general surgeon who is not really interested in the problem and who is being subjected to constant pressure from the superintendent to free the bed for better paying, less troublesome and more fluid patients to arrange for the transfer of this unwanted member of their professional family to the county hospital, the poorhouse or to some nursing home, even in the light of certain knowledge that this but signs his death warrant after subjecting him to a lingering, painful illness complicated by bed sores, kidney and bladder stones, renal infections, a constantly wet or soiled bed, spasms and deformity. Who can blame the family or the patient for believing that death cannot come too soon under such circumstances and that when it does come it is a merciful release to all concerned? This particular problem is so big that it is not only a matter for the attention of the trustees and staffs of small hospitals but should be the active concern of the big hospitals and the communities as well. The key to its solution still remains with the general surgeon, however. He can prevent the invalidism by increasing his knowledge of how to handle such injuries and he can make the community see that failure to provide means for the rehabilitation of these patients is not only short-sighted and uneconomic but, worst of all, is uncharitable and an evasion of proper responsibility as well.

What, then, are the responsibilities of the trustees and staffs of hospitals in relation to patients with injuries to the nervous system? If we consider the large hospitals first the answer to this question is easy. All large municipal hospitals, all large privately supported hospitals in large centers of population and all teaching hospitals, if they would fulfill their duties and

obligations to the public and to the medical profession can do no less than include in their organization a full fledged formal neurosurgical clinic. The details of organization of such a clinic are too well known to need recapitulation here but they must include a visiting and house staff of neurosurgical specialists and all ancillary services and personnel. Their function is to set the style in the treatment of injuries to the nervous system to contribute through public and professional educational activities and by investigation and the writing of significant papers to the advancement of the art, science and practice of medicine in its largest sense to teach under graduate and postgraduate courses and to act as a center for consultation and information, and as a court of last resort for the staffs of the small community hospitals. To this end the staffs of these larger hospitals must insist that all serious, undiagnosed difficult deteriorating and operable cases of injuries to the nervous system be transferred to them for definitive treatment. They must be prepared to visit and consult with the staffs of the small hospitals and they must do everything they can to discourage the doing of peripatetic neurological surgery. If they do operate under the necessarily unfavorable surroundings of a small hospital the operation must be strictly reserved for those occasions when it cannot possibly be avoided and because it is in the best interests of the patient. By the maintenance of such standards and the imposition of a high degree of professional integrity among themselves, these larger hospital specialized staffs will do more to encourage and strengthen the hands of the willing but overworked local general surgeon and improve his and the medical profession's public relations than any other means that is presently at hand.

The responsibilities of the trustees and staff of the small hospital are even more important to recognize and infinitely more difficult to meet. First the staff. The prime requisite for the staff is to discipline itself to a point where its members are constantly striving to increase their skill and knowledge improve their professional standing and, if they cannot lead at least remain in the front rank of the medical parade. This requires familiarity with the contents of the leading medical journals attendance at appropriate medical meetings visitations to other hospitals monthly critical analysis of failures mistakes and deaths good and up to date records and postgraduate education not only along those lines of basic science and general medicine and surgery that are appropriate for all who practice good medicine but also in such a way as to promote individual specialized knowledge so that each staff member knows more than any other member about some one surgical specialty. The essence of this knowledge is for each individual to know what he does not know about his special interest. He will therefore see that help is called for as soon as necessary and that if it is in the best interests of the patient to do so the latter will be promptly moved to a larger hospital with better facilities and a more specialized staff. Only in this way can the members of the staff be ready to meet the responsibilities that are thrown upon them with the admission to the hospital of patients who have been severely injured.

The meeting of these requirements is particularly important in the handling of traumatic neurosurgical problems. As has been pointed out above, most of such problems are first seen by the general surgeon in a small community hospital. If he is competent and familiar with his own necessary deficiencies and has, as he should have, a working knowledge of traumatic neurosurgery, he will promptly recognize that, after the preliminary treatment of surgical shock and the collection of enough evidence to warrant a presumptive diagnosis which predicates a condition the care of which is beyond his skills, such patients should be promptly transferred to a neurosurgical center. It is obviously in the best interests of the patient to do this as soon as possible. For this reason and because all the essential information has already been collected, he will not waste the patient's, the consultant's or his own time by asking the consultant to visit the local hospital to see the patient prior to transfer but will make all necessary arrangements over the telephone. This same attitude will and should govern in all serious undiagnosed difficult, deteriorating and operable neurosurgical conditions. The only exception to this rule occurs when, for some good reason, transportation to the neurosurgical clinic is impossible by reason of the breakdown of all facilities. It is at this point that it is of value to have one member of the local surgical staff trained to a greater extent than the others in this specialty. He should, if he has to do so, be able to carry out life-saving emergency cranial or spinal surgery. This is not done as a preferred method of treatment but as one that is better than none at all which, because of the breakdown of transportation, is the only alternative. The simple neurosurgical cases that are not in the above category and that stay simple can be treated as well locally as they can be in a special clinic provided the surgeon treating them has had the necessary postgraduate education, knows his own limitations and is willing and eager to get help whenever there is any question of his needing it.

Neurosurgery done in the small community hospitals by specialists who travel over the country operating in one such hospital after another is bad neurosurgery. It is bad for the local doctor because he has to accept more of the responsibility for preoperative diagnosis and preparation, and for postoperative care than he is able trained or should be expected to. It is bad for the peripatetic consultant because it is a deliberate performance that is contrary to all his training, is a sacrifice of his ideals and an affront to his knowledge of what is best for the patient. It is bad for the patient because it deprives him of his proper right to survival and complete recovery without affording him any opportunity to object to this deprivation. Because such surgery is more convenient for the local doctor, the consultant, the patient's family or his employer is not an adequate reason for its performance. Proper neurosurgery, the kind of neurosurgery that every doctor would demand for his wife or child, can only be done in an adequately equipped neurosurgical operating room by a properly trained neurosurgeon, assisted by specially trained operating nurses and residents and having immediately available such facilities as a blood bank and

other special consultants. In addition the neurosurgeon must personally or through his resident live with his patients twenty-four hours a day. Just because a patient does not happen to be a member of the doctor's family is no reason to deprive him of facilities that would be available otherwise.

No staff can meet these requirements without help and encouragement from the trustees and support from the community. It is for the trustees, acting as representatives of the community, to see that such support is forthcoming and to provide by proper supervision and the wise expenditure of the funds entrusted to them, the necessary equipment and ancillary personnel as practical evidences of their encouragement. In addition they should manifest their practical interest in the patient's welfare by attendance at the monthly clinico-pathological and record meetings, by encouraging postmortem examinations, by promoting the doing of good pathology, by seeing that the records are adequate and up to date, by providing opportunity for postgraduate education of the staff and by the maintenance of professional discipline. All these requirements and others are part of what the trustees owe to the community in return for the money that has been raised and given to them to maintain the hospital. Because of their position they can wield a tremendous influence both on the community and on the practice of medicine in it. If they tacitly approve the reprehensible actions of a staff member by failing to discipline him on account of inadequately explained deaths, invalidism or infection, for example, they merely depress the level of community medical practice, promote the doing of further bad medicine and are false to the trust imposed on them by the citizens. If, on the other hand, a doctor such as referred to above is deprived of the right to use the hospital on account of his actions, then the practice of medicine in the community is improved, individual patients learn that their interests are being protected, the delinquent doctor is forced to mend his ways and increase his knowledge or retire from practice and the public, seeing that their money is being expended wisely and in their best interests, gladly support a bigger and better hospital. This in turn leads to the acquisition of a pathologist, a better x-ray man, an anesthetist, a more skilled laboratory technician, a physiotherapist and so forth, all of which leads to a further improvement in medical practice and hence better care of the individual citizen. The trustees' power is great—they can make or break a doctor locally by withholding the privileges of the hospital from him or not. They should use such power with discretion but once they have been satisfied that its exercise is in the best interests of the community they should use it promptly, efficiently and without fear or favor.

Traumatic neurosurgery is no longer the sole business of the specialist in his cluttered halls. It is a charge on the general surgeon along with acute abdomens, fractures of the extremities, amputations and the like. The general surgeon cannot properly meet this responsibility without help from the specialist, however, and must so organize his small community hospital as to take the best advantage of the nearest neurosurgical clinic. The trustees of his hospital will support this arrangement, serve as a liaison between him

and the public he treats and which supports him and enforce proper self discipline in his own best interests. The specialists and the neurosurgical clinics in their turn *set the standard of practice of neurosurgery* guard the local surgeon against the depredations of the peripatetic specialist, provide active consultative support and co-operation through facilitation of transfer of appropriate patients to the large surgical centers, and aid the general surgeon to acquire greater knowledge, increased efficiency and a truer appreciation of his own importance in the community by seeing to it that he has every opportunity to participate in graduate and postgraduate education.

Rehabilitation

Rehabilitation is a poorly understood and a widely misused word. *Webster's Collegiate Dictionary* gives as a fourth definition of the verb "To fit to make one's livelihood again." Our modern way of life is so complicated that, while this is undoubtedly desirable it has to be regarded as a secondary goal the first being physical and psychologic rehabilitation. According to the *Shorter Oxford English Dictionary* it means "The act of restoring to a previous condition to set up again in proper condition." The usual attempts at rehabilitation arise out of a desire to "restore to a previous condition" but since most of the patients who need rehabilitation after their injury have permanent organic functional defects that make such a restoration impossible, the attempts are bound to fail. As a result the patient becomes impatient, discouraged and unco-operative. If on the other hand, the alternate definition—"to set up again in a proper condition"—is used as the basis of rehabilitation then it is apparent that such a "restoration to a previous condition," in addition to being impossible is not necessary. Thus the first requisite for successful rehabilitation is to set one's goal within the practical limits of success.

Setting one's goal for rehabilitation within the practical limits of success must not mean that once these limits have been established they cannot be expanded. The limits of accomplishment must be regarded as elastic. This ability to expand is inherent in the rehabilitation of all those in need of it, but particularly so in the group of patients that have nervous-system injuries. In these individuals the learning of one skill leads directly to the learning of another that had previously been regarded as beyond the realm of accomplishment. A quadriplegic whose only talent appears to be limited to the acquisition of the ability to sit erect in bed has by that very ability started the process that eventually enables him to wash and feed himself to develop hitherto unusable arm muscles, to propel himself in a wheel chair to use a typewriter to visit neighbors and friends to become a member of the local social community again to redevelop a co-operative cheerful attitude toward family friends and community and eventually—just because he took the trouble to learn to sit erect—to become an asset instead of a liability to the community. The second requisite for rehabilitation is never to be satisfied with what you have.

The third requisite for rehabilitation is to realize that true rehabilitation

means rehabilitation of the whole and not just of isolated parts. No man or woman can exist as a mass of unconnected isolated parts or skills. This is especially true when an injury has deprived us of the ability to use some of the skills that we have previously taken for granted. Just as in the normal individual all physical, intellectual, emotional and social activities must be integrated to be effective and to keep the individual on an even keel, so the invalid in need of rehabilitation must reintegrate his damaged machine so that it acts as a unit, even though the unit is no longer as effective as it was before the injury. Since total rehabilitation is made up of the sum of the rehabilitation of the various units as well as of their integration, neglect of any one unit will have a deleterious effect on the rehabilitation of all others and of the whole as well. It is useless in a patient who has sustained a craniocerebral injury for example, to expect effective rehabilitation if his headaches are treated but his declining general health and the deterioration that goes with it are allowed to continue unchanged. It is inevitable that such a patient will remain and prefer to remain an invalid at home that any physical effort will lead to fatigue and that a permanent neurosis with sufficient subjective symptoms to justify it can be predicted and will inevitably develop instead of the desired rehabilitation. An even better illustration is found in the confusion that has arisen over the need for paraplegics to learn to ambulate as a part of their rehabilitation. The ability to ambulate is only one unit of the greater total problem of the rehabilitation of such a patient. Another is the ability to control his bladder and bowel, another is the ability to take full care of himself, another is the maintenance of reciprocally pleasant relationships with his family, friends and community and still another is to have an effective knowledge of himself, an insight into his own motives and a realistic appraisal of his deficits. Not only must his physical capacities be developed to the utmost but his intellectual capacity as well.

The need for learning ambulation has been much discussed. My experience would indicate that learning to ambulate derives its importance among the other skills of which it is one from the fact that it is the first major test that the patient must successfully negotiate if he is to be able to face up to the greater and more searching demands made on him later. It requires both physical and mental adjustment and development, and its accomplishment carries with it a tremendous lift toward further efforts. It serves also as a measure (but not the only measure) of the individual's determination to remain rehabilitated. The practical use of continued ambulation is extremely limited as such, even to a paraplegic, but the continued ability through use and practice, to move about freely with the aid of braces and crutches sets a continuing ever-present standard of accomplishment that keeps the other more important rehabilitatory attributes at an equally high level. Nothing is more discouraging than to see a patient who has left the hospital proud of his ability to do a good swing-through, determined to "get some sort of a job," who has bladder and bowel control, a good emotional and intellectual understanding of his own problem and a good familial and community

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lationship stop ambulation because it was too much trouble" majority this is the first step toward disintegration with its use of rumption of enemas, a cynical attitude about employers and the in employability family quarrels, the abandonment of all social and the reinstitution of the condition everybody had worked so hard at. The third requirement for rehabilitation then is that not each part of the organism be rehabilitated but that the whole man combination has to be "set up again in proper condition" and kept that way. The fourth requirement for rehabilitation is that the patient's condition all have been correctly diagnosed the pathology correctly understood and adequate treatment provided, and any organic disease if present be static and have reached an end point prior to the start of the preliminary steps of rehabilitation. These requirements are obvious that they are frequently overlooked and consequently not followed. The following illustrations will serve to emphasize these points. It is for example, that a patient who complains of pain and other symptoms in his right hand sufficient to prevent gainful employment for months after minor injury to the same forearm will be classed much more of a neurotic or a malingerer than as legitimately suffering from what is correctable Homans' Minor Causalgia. On investigation it will often appear that none of the doctors who have cared for the patient has even thought of this diagnosis. Again, it is useless to try and tell a patient that his back no longer hurts following removal of an intervertebral nucleus pulposus, that any associated bladder disability is traceable to chronic prostatitis and that he should return to work despite these symptoms, when adequate investigation demonstrates that the patient has an adhesive cauda equinitis with partial denervation and that his bladder as well as perineal hyperaesthesia and pain down both legs are unrecognized postoperative subarachnoid hemorrhage or periradicular irritation. An explanation for constant headaches and dizzy attacks associated with inability to work, that is based on a diagnosis of post-traumatic neurosis cannot be expected to hold water when proper investigation demonstrates the presence of a cerebral subdural hematoma that has been unrecognized and even unthought of since the accident and the craniotomy which offers the only possibility of clearing up the patient's symptoms. The inability of a paraplegic patient to learn to balance to care for himself to ambulate and the consequent failure of all efforts to rehabilitate him are immediately explainable when the jack-knifing that has persisted as a part of his spasms have been "relieved" by obturator neurectomy. The fact that peripheral operations has at last been recognized. With proper denervation that includes the paraspinal muscles the patient promptly learns to walk, including ambulation that have been denied him because of incorrect diagnosis and thus can be rehabilitated with relative ease. A similar patient attempts rehabilitation but refuses to let the doctor stop his spasm by any surgical procedure. In consequence his self-caused ineffective, he cannot move himself about freely he cannot balance in

cannot ambulate his bladder cannot be trained and shrinks, and his braces must be so designed as to exert corrective pressure on his spastic legs. The acquisition of these various skills and others is out of the question until the spasms are eliminated. Moreover his complete rehabilitation may never be possible because of the permanent organic changes that have developed. Rehabilitation predicates the best and the most painstaking accuracy in medical and surgical diagnosis and care. Without these prerequisites rehabilitation is impossible.

The fifth requirement for rehabilitation is that no attempt be made to remake the individual to a pattern that is different from his former pre-accident one. The ordinary common laborer cannot be expected to learn watchmaking, for example just as there is little use in providing a correspondence-school course in accounting for a patient who has had no more than one year in high school. This is all self-evident but it carries with it implications that are not generally recognized. Patients who have permanent organic and functional deficits that are the result of their injury and which make it impossible for them to return to their old occupations must not be cast aside without any alternative employment on that account. Every effort should be made to place them in a position where they can work in a job that is similar to their old one but that is within their new capacities. This is not as simple as it appears. Let us take, for example, the patient who has ruptured an intervertebral disc in industry has had the extruded nucleus successfully removed and the radiculitis relieved. He comes to the end of his convalescence and is ready to go back to work. He is told and has demonstrated to himself that he can do any kind of work that does not involve lifting with his back. His previous job had been that of an iron-moulder and was one that required the lifting and handling of heavy plates of steel. It is obvious that he cannot return to this work. However he knows no other. He goes back to his previous place of employment and asks for re-employment but in another job. The usual answer is (a) that there is no other job open but to come back some other time (b) that he is unemployable because he is uninsurable and (c) that his employment in another job in the plant involves disruption of the seniority relationships in the new job and that the union will not consent to it. He then goes from employer to employer and each time receives what amount to the same answers as soon as he says that he has had a back injury and an operation. If he tries to conceal these facts the information is available to and is obtained by all major employers and by their insurance staffs from a central listing of industrial injuries. The patient shortly becomes convinced that he is unemployable develops new symptoms as well as a persecution complex to rationalize his unemployability and soon becomes *actually* unemployable on this account. Rehabilitation should prevent this and is impossible in the face of it. Rehabilitation predicates not only that the process be governed by the patient's pre-injury make-up characteristics and abilities but that steps be taken to see that re-employment even if it cannot conform to the pre-injury standards, is nevertheless provided and is as like the latter as possible.

Finally the most important requirement of all for rehabilitation is that the person to be rehabilitated shall be fired with the desire to be independent—*independent physically independent intellectually and emotionally independent socially and if possible to have some degree of independence financially*. He must also recognize that true rehabilitation is impossible as long as charity that is undeserved even though given under the name of a pension a compensation payment free tickets to entertainments, or unearned community privileges is accepted. It is still charity and under such circumstances is as disintegrating to the soul of the recipient after an accident as it was before. This does not mean that the true charity that is well deserved is bad as long as it is recognized as charity and as long as it is deserved. It does mean however that no matter what else is accomplished in the field of rehabilitation real rehabilitation has not been attained as long as undeserved charity is considered to be the recipient's right. Without this drive to be independent, attempts at rehabilitation are no more than empty gestures and a waste of time. All other requirements can be met but without the active, intelligent determination of the patient to make the most of what he has or can develop to become a respected, self respecting member of his family and his community and to be as independent as possible from every point of view including the physical, all efforts at rehabilitation will fail dismally.

It must be evident that rehabilitation to be complete in any one individual, requires a highly complex education which has to cover the ground from the practical matter of job training to the successful institution of such ephemeral psychological relationships as those that must exist between a man and his wife and his community. The very essence of rehabilitation however rests within the patient himself and is compounded of his determination to make the most out of *all* his potentialities and to face courageously and to conquer the multiplicity of educational hurdles that must be topped to attain this desired end. The most important of these hurdles are physical emotional psychological sexual and financial. The final goal is reached when the victim has developed sufficient insight about and understanding of his own deficiencies to grasp the significance of these barriers, to be able to laugh at his failures and not get puffed up at his successes and to refuse to admit that he cannot improve his condition no matter what degree of facility he may have already attained.

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